DRAFT FOR COMMENT

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

VOLUME 1

OVERVIEW REPORT AND RECOMMENDATIONS

by

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Draft for Comment

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

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OVERVIEW SUMMARY

A total of 17 individual expert reports were obtained on a variety of issues bearing on the safety and quality of Edmonton's drinking water. These reports provided the assessments of specialist experts from Germany, the United States and Canada. In all cases but one, the experts visited Edmonton and were able to provide their views based upon direct observations and inquiry as well as written documentation. The experts selected for this study represent leaders in their specific fields and their views represent state-of-the-art knowledge.

These expert reports provide the basis for this overview assessment. The major issues covered included: raw water quality, monitoring procedures, treatment practice, treated water quality, health and aesthetic considerations, and options for improvement.

The study concludes that the North Saskatchewan River, as it arrives in Edmonton, provides a good source of raw water at most times of the year. The raw water quality at the center of Edmonton, where the Rossdale intake is located, is adversely influenced by contamination from upstream storm sewers. This is not a good location for a raw water intake and it should be relocated to obtain water quality equivalent to that available at the upstream E.L.Smith plant. Reliance on being able to normally meet guideline levels for treated water quality is not a sound basis for overlooking the documented problems with the Rossdale raw water source.

The water quality monitoring capabilities of the Edmonton water laboratory were reviewed and some deficiencies were found. Specific recommendations were provided to improve laboratory efficiency and capability to meet current and anticipated needs in water quality monitoring.

Current treatment practice was reviewed and compared to practice elsewhere in Canada, the United States and Europe. Processes were generally found to be equivalent to those used elsewhere in similar circumstances, but considerable scope was identified for operating changes which could be initiated by a process improvement team. Opportunities exist for better and more reliable treatment performance and more cost-effective chemical use. Likewise, given adequate devotion to characterizing the probable causes, sources and distribution of taste and odour problems, there is considerable scope for controlling such problems in the future without necessarily adding major new treatment processes.

Review of treated water quality relative to the license requirements indicates that Edmonton's record of compliance in recent years has been excellent. Comparison of treated water quality for normal conditions with survey data from other cities indicated that Edmonton's finished quality is comparable. These comparisons could not reflect consideration of problem times, such as spring runoff, because data for problem events was not available for other cities. However, taste and odour problems of some type do occur with most water utilities.

Evaluation of health concerns indicated that there was no cause for concerns associated with substances covered by the Canadian Drinking Water Guidelines or for additional identified substances for which adequate toxicological testing has been done. Less certainty can be addressed to those substances which have not been subjected to extensive toxicological testing or which cannot be analyzed by our current techniques. However, even considering these components, there was little objective reason for health concerns associated with trace substances in Edmonton water.

There were concerns expressed over the potential health problems which could arise from inadequate control of disinfection. On one extreme, inadequate disinfection could allow the passage of infectious microorganisms in the treated water. On the other extreme, overzealous disinfection could leave unacceptable concentrations of the disinfectant chemical or its byproducts in the water. Development of site specific disinfection criteria for Edmonton will allow selection of an appropriate balance between the extremes. Relocation of the Rossdale intake away from likely sources of contamination together with improved process control and monitoring will also contribute towards lessening concerns associated with disinfection.

Options which can be pursued to achieve these improvements were reviewed and several recommendations for specific action have been made. The most critical recommendations call for relocation of the Rossdale raw water intake to avoid impact from upstream storm sewer discharges, establishment of an in-house process improvement team to maximize the beneficial operation of existing treatment processes, and improvements in water laboratory operations and capabilities to insure water quality control under all forseeable conditions.

1. STATEMENT OF NEED

Edmonton derives its raw water supply from the North Saskatchewan River (Figure 1). This water source is subject to variable quality inflows and seasonal conditions which cause fluctuating raw water quality. In particular, spring runoff and major storm events have led to aesthetic problems with the treated water distributed to Edmonton consumers (noxious taste, odour and colour). These problems along with an increased public and professional concern regarding possible links between trace substance intake and human health have caused many questions to be raised about the quality and safety of Edmonton's drinking water. As well, concerns with possible waterborne transmission of disease by the presence of pathogenic microorganisms have been expressed. These circumstances, over the past several years, have caused many Edmontonians to doubt the quality and safety of their drinking water.

The raw water source and drinking water supply in Edmonton are subjected to extensive monitoring on a routine basis. In addition, many special studies have been conducted on the North Saskatchewan River and the treated water supply to address specific concerns. While all of the information which has been gathered may contribute to understanding specific aspects of the problem, concerns with the quality and safety of Edmonton drinking water persist. Consequently, there is a need for an independent, critical and comprehensive review of Edmonton's drinking water to inform consumers about how our drinking water compares with drinking water in other jurisdictions, whether there are valid health concerns which must be addressed and whether there are feasible solutions to the intermittent aesthetic problems which have occurred.

The purpose of this report is to provide consumers of Edmonton's drinking water with a thorough, independent, expert assessment of the quality and safety of their water supply. The study terms of reference are provided in Appendix A.

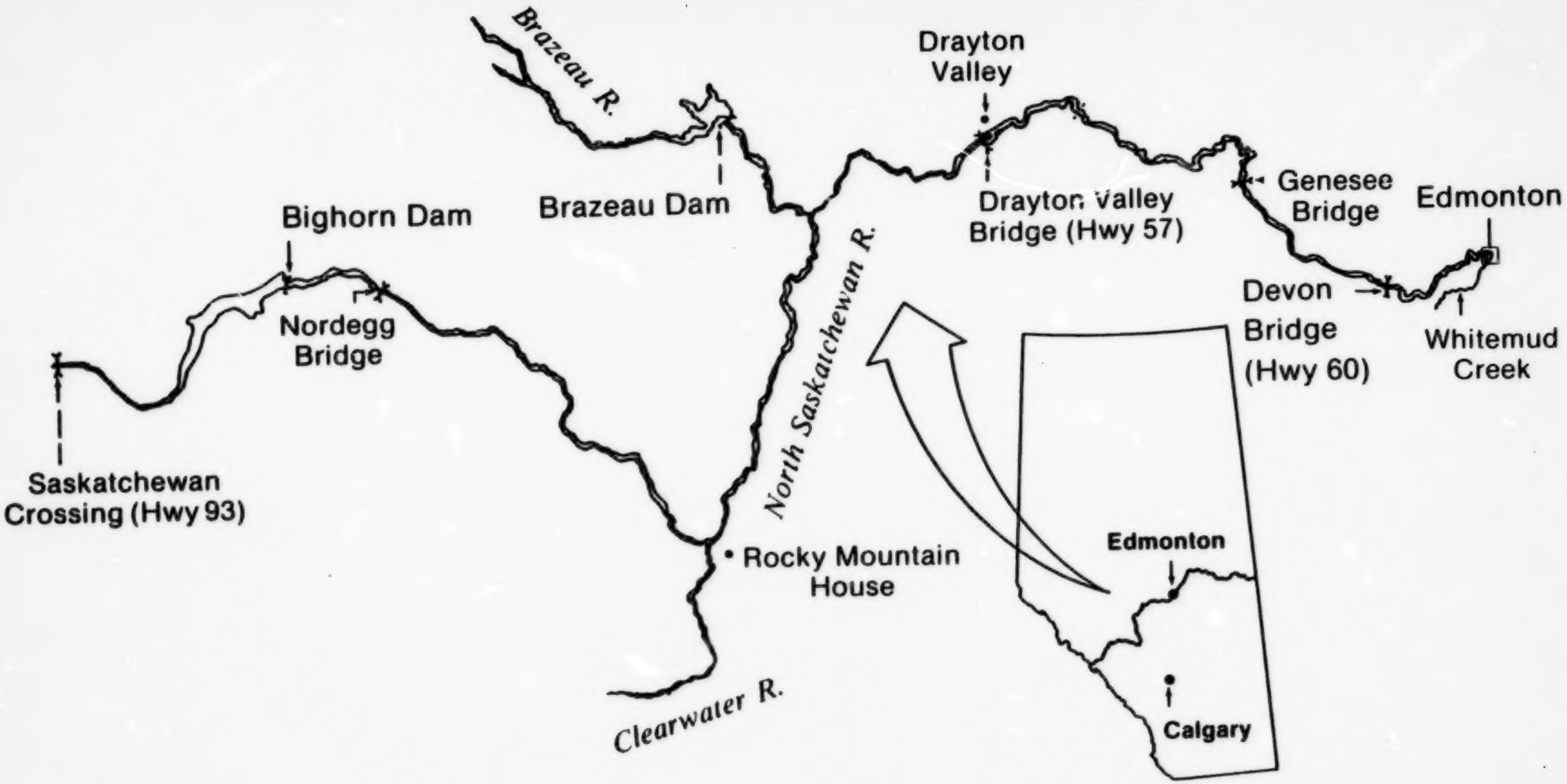

Figure 1. The North Saskatchewan River Basin Upstream of Edmonton.

2. INTRODUCTION

2.1 General Issues

Confidence in the safety and quality of our water supply must be based on thorough, critical and continuing assessment of available knowledge. That confidence will only be assured by maintaining open access to all relevant information about the quality and safety of our water supply. The City of Edmonton should be commended for exposing the water treatment and supply system to this critical review. A willingness to undergo this type of critique is not generally prevalent in the water utility field. The scope and scale of this inquiry may be unique. Hopefully the City of Edmonton will find enough benefit from this exercise to justify continuation of an open door policy with its consumers.

Water treatment practitioners have been criticised by some as having not developed anything new in 50 or more years. There is an element of truth to this accusation, in that the fundamental water treatment processes of coagulation and filtration, for particle removal, and disinfection, for inactivation of disease-causing organisms, have been known since the turn of the century. Applying these processes in the same way that they were used 50 years ago can, in most cases, convert an unsafe surface water source into a potable water. Refinements in performance and specific quality improvements can be achieved by applying newer technologies and practice, but the basic transition from an unsafe surface water to a finished water unlikely to cause waterborne disease epidemics can, in most cases, be achieved without much departure from classical practice.

The robust capability of conventional treatment processes has led some to believe that water treatment is essentially easy and undeserving of any sophisticated engineering improvement. With outdated knowledge and limited talent, it may be possible to treat water to a marginal potable quality. However, cost-effectively treating water from a variable quality source to produce a finished water which will consistently meet current quality expectations is not easy. In fact, given some of the challenges offered by the vagaries of the North Saskatchewan River, consistent, effective treatment in Edmonton can be very difficult at times. Hence, the challenges offered to the Edmonton water treatment plants should not be underestimated.

Achieving reasonable performance goals requires maintaining a healthy respect for the degree of difficulty that must be faced, combined with a determination to produce finished quality which Edmontonians can regard as superior. Given the treatment challenges which must be met in treating a variable river source, adopting a target of marginally adequate quality would only insure that unacceptable quality will occur too often.

Unfortunately there is no National water treatment research laboratory, such as currently exists for wastewater research (Environment Canada Wastewater Technology Centre at Burlington, Ontario). This deficiency leaves individual municipalities without any national technical support for solving problems which may arise.

Dealing with the existing circumstances, one must realize that an assessment of drinking water quality, and its safety, is more than a narrow scientific and technical inquiry which can engage in absolute truths and ultimately provide black and white answers. In reality, judgement and common sense often must play a larger role than the limited, reliable, scientific knowledge which may be available. The accomplishments of our modern technology are often regarded as the result of an absolute and precise understanding of all the underlying natural phenomena. While this may be the case in some circumstances, in general, our thorough knowledge of what makes things happen is often overwhelmed by what we do not know. This circumstance becomes more relevant the more we seek to control and improve the quality of our lives by eliminating progressively less prevalent causes of illness. The pursuit of drinking water quality which we can be assured will have no adverse health effects over a lifetime of consumption represents such a challenge.

We know with a high degree of certainty how to prevent the waterborne transmission of diseases such as typhoid or cholera, which once killed thousands of people in epidemics and which still cause waterborne disease and death in under-developed countries. In contrast, we cannot even be certain that the trace levels of chloroform, which are commonly present in drinking water, can actually cause cancer in humans, let alone be certain about what levels would be necessary to cause a particular rate of disease occurrence.

Faced with such uncertainty, scientists who must recommend safe levels of chemical substances have necessarily responded with reasonable, but necessarily pessimistic assumptions about the likelihood of harm. Furthermore, scientists will apply safety factors to those assumptions to compensate for variations in exposure, human population sensitivity and the inevitable uncertainty of translating safe levels from controlled experiments (usually with laboratory animals) to general population exposure. These conservative measures are necessary expedients to allow us to apply the knowledge that we do have. Most decisions about safe levels would require much better and more comprehensive knowledge if we tried to make very precise recommendations about the transition from safe to unsafe exposure. In other words, the abiding principle when deciding on safe levels of substances in drinking water, given knowledge which is usually limited, is to err on the side of caution. That is a prudent approach and one which should not be abandoned without substantial justification.

2.2 Edmonton Water System

Edmonton's drinking water is produced from two major facilities, Rossdale and E.L. Smith. The first is located northeast of the 105 Street bridge and the second is across the river and southwest from Terwillegar Park (Figure 2). Four individual water treatment plants are located at Edmonton's two water treatment facilities. Plant #1 was commissioned at Rossdale in 1947, followed by Plant #2 in 1956. These are essentially equivalent plants in design and layout. Plant #3 was added in 1967. Finally, the E.L. Smith plant, designated plant #4, was opened in 1976.

Through staged upgrading at the older plants they are all now using the following processes: alum coagulation, clarification, chlorine dioxide disinfection, fluoridation, rapid sand (dual media) filtration and chloramine disinfection. Partial softening with lime and recarbonation is practiced at plants 1,2 and 4. Finally, powdered activated carbon can be fed at the point of alum addition, as needed, for organics removal. These processes would qualify as complete treatment according to the Canadian Drinking Water Guidelines.[10] Auxiliary treatment, in the form of pre-chlorination, pre sedimentation and post-chlorination are not practiced in Edmonton. However, the powdered activated carbon and possibly the chorine dioxide could be interpreted as "Other advanced forms of treatment approved by the control agency may be considered equivalent to any

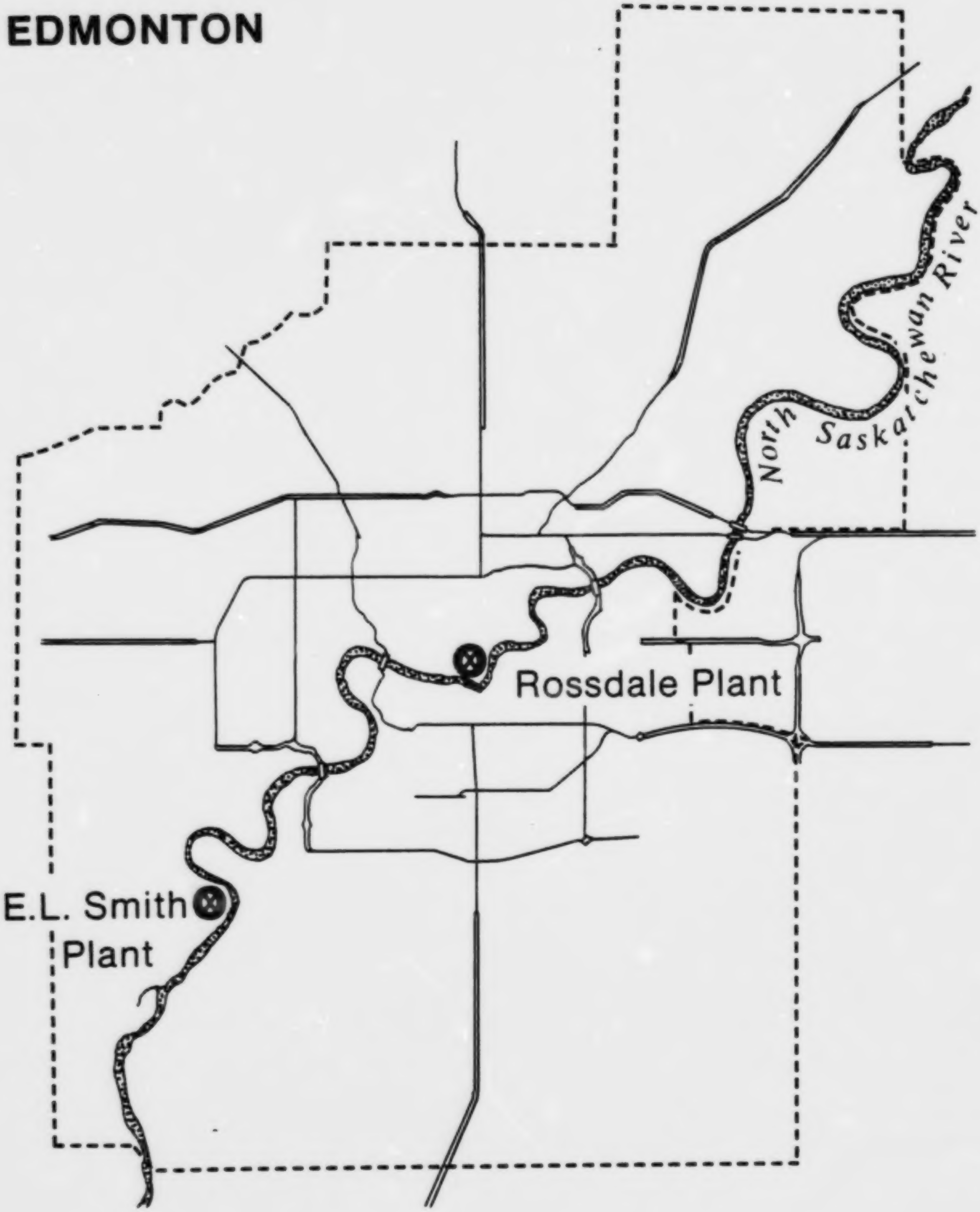

Figure 2. Location of Edmonton Water Treatment Plants.

of the traditional methods named above.", in accordance with the Canadian Drinking Water Guidelines.[1]

3. PUBLIC INPUT AND REVIEW

The study was effectively initiated after Edmonton City Council approved a bylaw authorizing a joint agreement with Alberta Environment to fund the inquiry on November 12, 1985. Newspaper advertisements were placed on Saturday and Sunday, November 23, 24, 1985, announcing the study and soliciting input from the public. As a result of these advertisements, a total of 61 written responses were received and acknowledged. The terms of reference for individual experts were adjusted, as much as possible, to include issues raised by the public response. A toxicologist was assigned to deal specifically with a few health-related issues that were raised but did not fit with any prior expert assignment.

A public opinion survey was also commissioned to assess the attitudes and concerns of Edmontonians. This was performed by MTK Research Associates using 400 randomly selected telephone interviews in June, 1986. Detailed results are provided in Volume 2.

A few interesting results include:

1. Of people surveyed, 84.9% usually drink Edmonton tap water.
2. Overall 15.6% were very concerned and 28.2% were quite concerned about the quality of Edmonton's water.
3. In contrast, 33.6% were not too concerned and 21.9% were not at all concerned.
4. Respondents rated Edmonton Water and Sanitation's performance at, 5.4% excellent, 43.1% good, 30.4% fair and 11.2% poor.
5. Of all respondents, 13.4% believe they have become sick from drinking Edmonton tap water.
6. Of all respondents, 75.9% believe it is possible to make Edmonton's drinking water safer than it is now.

Following general release, the public will be invited to comment on this draft report and a public review meeting will be held in the fall to solicit final commentary on this report. In the meantime, two experts who have had no previous involvement with this study have also been retained to critically review the draft.

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When all review comments have been received, this report will be revised, as necessary, to address the comments and the final report will be prepared for submission.

4. THE STUDY PROCESS AND EXPERT PANEL

An investigation as broad as outlined in the Terms of Reference for this study requires a multi-disciplinary approach. Given the range of issues which had to be addressed, no individual could be truly expert in all the relevant areas. The public may see scientists and engineers as a group of technocrats who should all be able to converse and freely exchange knowledge on a common ground. In reality, science and technology have become extremely specialized. This makes it very difficult for any individual to be truly expert in more than a very narrow specialization. Those claiming to have broad expertise in a wide range of issues are necessarily limited to superficial knowledge in the individual specialities.

The need for a thorough evaluation of Edmonton's drinking water situation dictated that the study team be assembled from a large number of specialized experts rather than from a smaller group of generalists. This approach required considerable coordination to assure that the overall Terms of Reference would be successfully addressed in an integrated fashion.

The experts selected for this study represent leaders in their specific fields and their views represent state-of-the-art knowledge. Brief descriptions of their specific backgrounds are provided in Appendix B. In all cases but one, the experts visited Edmonton and were able to provide their views based upon direct observations and inquiry as well as written documentation.

This Overview Report seeks to integrate the individual expert reports into a meaningful assessment and a useable plan for action. Although all the expert recommendations are commended for consideration by Edmonton Water and Sanitation, Alberta Environment and the Edmonton Board of Health emphasis has been placed on some recommendations which are judged to be essential for addressing the major issues. Those recommendations will be presented in this Overview Report.

In a small number of cases, the experts presented conflicting views. In such cases, if the recommendations were ones which met the above criterion for inclusion in the Overview Report, I reviewed the justification provided in support of the conflicting recommendations and the relevant qualifications and experience of the respective experts. With this basis, a selection or a compromise recommendation was brought forward to this Overview Report.

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5. QUALITY OF THE RAW WATER SUPPLY

5.1 Comparison with Surface Water Quality Criteria

The quality of the North Saskatchewan River, as it arrives in Edmonton is good for a surface raw water source. Comparison of average raw water quality with the Alberta Surface Water Quality Objectives (or the most stringent objectives from other jurisdictions where the ASWQO were higher or non-existent) indicates compliance for everything except turbidity and phenolics. The detailed water quality review is provided in the Hrudey-1 report (Volume 2). This review indicated that there is a need to update the Alberta Surface Water Quality Objectives for a few parameters, but these objectives are otherwise consistent with practice elsewhere.

The turbidity comparison was based upon Manitoba objectives which were set for raw water sources intended to be used for drinking water following treatment by disinfection only. There is no source which Edmonton could consider which would allow distribution after only disinfection. However, turbidity fluctuations are a significant challenge which must be met in treating water from the North Saskatchewan River. Low turbidity values in winter provide too little suspended matter to coagulate, flocculate and settle effectively with conventional chemical treatment. Spring and summer peak values can severely challenge the ability of the coagulation and filtration processes to achieve adequately clear water.

The phenolics are more difficult to interpret. The data chosen for this comparison were obtained at Devon between 1982 and 1986. The values measured for phenolics were higher than normally reported at the E.L. Smith plant, down river. In any case, the phenolics which are routinely measured by the standard colorimetric analysis method have not been shown to be equivalent to the specific phenolic compounds which cause problems in water supplies (for example, taste and odour). Because natural humic materials present in river water can give rise to phenolic structures, the significance of low concentrations measured by the routine colorimetric method is not clear. When more sophisticated analytical methods are used on the river water, they have usually failed to show signficant quantities of the specific phenolic compounds which are known to cause difficulty in a water supply. Hence, there may be a chronic problem with low levels of naturally derived phenolic substances occasionally in the river, but the importance of this to the quality of Edmonton's water supply has not been documented.

Iron and manganese values occasionally exceeded the Alberta Surface Water Quality Objectives. These two parameters are set strictly on aesthetic rather than health grounds and they pose no particular difficulty for removal by Edmonton's water treatment plants.

A single high value for raw water cadmium (out of 30 samples from 1982 to 1986) exceeded the Manitoba objective for water which could be distributed after only disinfection. The apparent high value occurred in 1982 and results have been consistently below detection limits (0.001 mg/L) since 1983. The latter data is consistent with data observed at the E.L. Smith plant .

The maximum value for nickel was 0.04 mg/L, again measured in one sample in 1982. Typical values since 1982 have all been below 0.012 mg/L, generally being less than 0.005 mg/L. The raw water objective of 0.015 mg/L was a 1980 U.S.Environmental Protection Agency proposal which was not reflected in their drinking water regulatory proposals in 1985 because nickel was not judged to require regulation based on current information.

The microbiological quality of the North Saskatchewan River, as it arrives in Edmonton, was assessed by Dr. Smith (report in Volume 2) and judged to be good. The same could not be said for microbiological quality of the river at the point of the Rossdale water intake, just east of the 105 St. Bridge. Raw water quality measured by microbiological indicator organisms was frequently poor because of the influence of storm sewer outfalls (85 in all) located upstream of the water intake.

Other adverse effects of the urban runoff were also documented (Hrudey-1 report, Volume 2). Large fluctuations in turbidity, chlorides, dissolved organic carbon, extractable organics and purgeable organics were found to occur at the Rossdale plant but not at the upstream E.L. Smith plant. The extractable and purgeable organics include hydrocarbons resulting from urban runoff. These differences, measured during the 1986 spring runoff, are illustrated in Figures 3 and 4.

There was no evidence of any adverse water quality impact from agricultural chemical usage or upstream municipal or industrial activity. Upstream water quality is adversely affected during spring runoff by high values of dissolved organic carbon and turbidity. In the spring of 1986, a dramatic rise in dissolved organic matter (Figure 5) occurred on March 1, causing a sharp rise in water color, disinfectant demand and associated treatment problems. This organic matter is

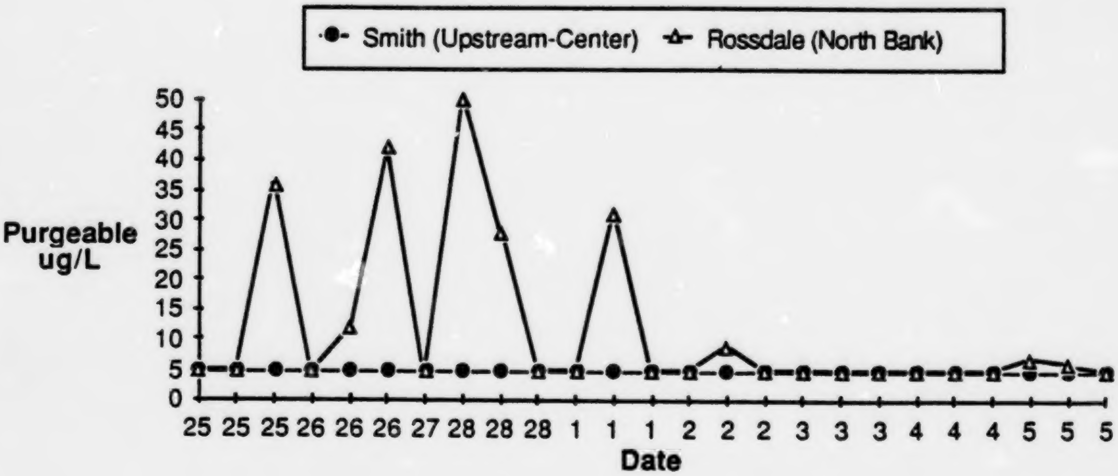

Figure 3. Comparison of E.L. Smith and Rossdale Raw Water for Purgeable Organics (February 25 to March 5, 1986).

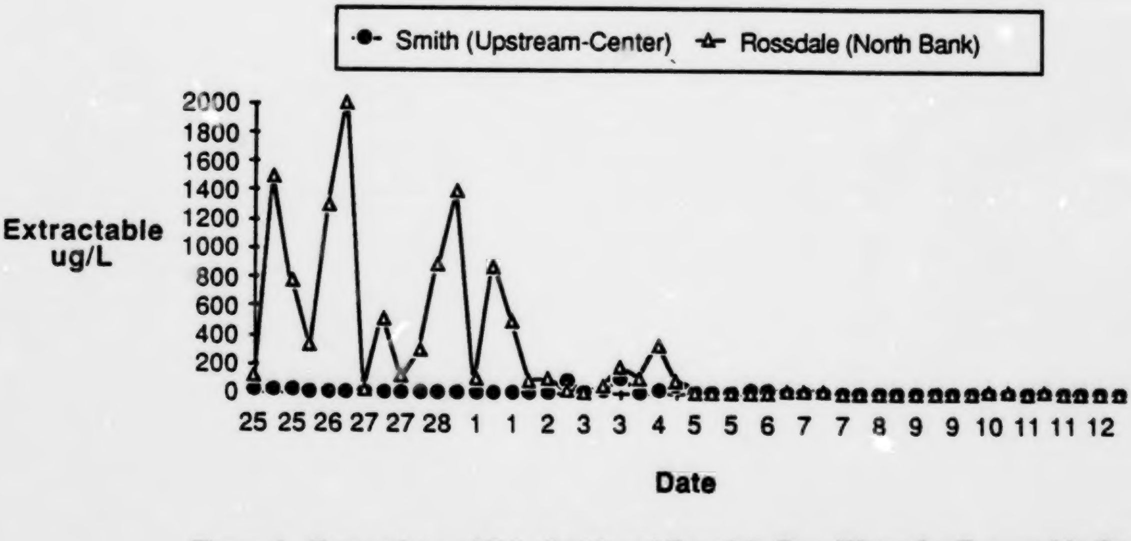

Figure 4. Comparison of E.L. Smith and Rossdale Raw Water for Extractable Organics (February 25 to March 12, 1986).

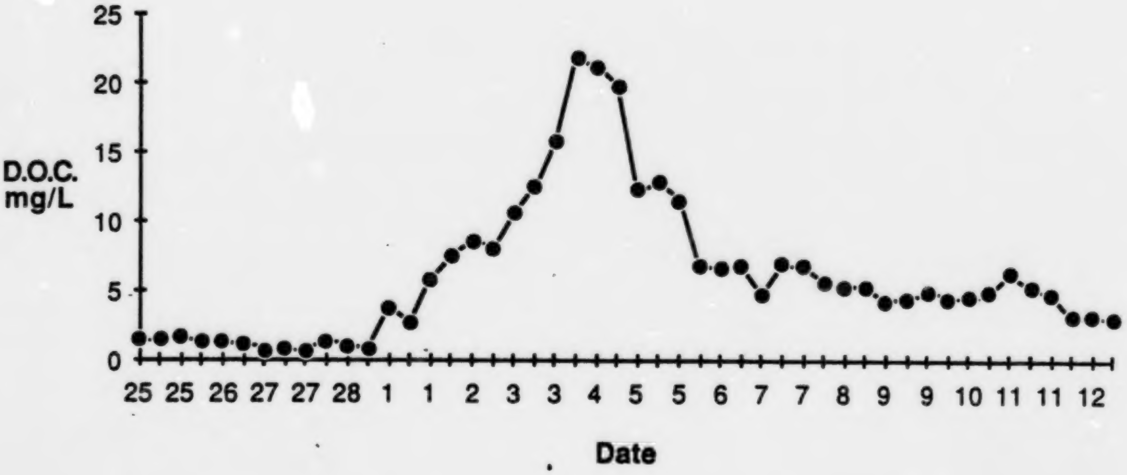

Figure 5. North Saskatchewan River Dissolved Organic Carbon Peak - 1986, from Upstream Rural Runoff.

believed to arise from snow melt leaching of decomposing vegetation as well as muskeg drainage from the upstream rural watershed

Although the raw water quality of the North Saskatchewan River entering Edmonton is generally good in comparison with water quality objectives, the effects of urban runoff periodically degrade the microbiological and chemical quality of the raw water source once the river has reached the Rossdale intake. The upstream rural runoff creates water quality conditions which pose significant treatment challenges but the inferior water quality from rural runoff does not carry the known microbial and hydrocarbon contamination arising from urban runoff.

5.2 Comparison with Raw Water Sources Elsewhere

The North Saskatchewan River upstream of Edmonton was compared with raw water sources for Ottawa, Brantford, St. Louis County (Missouri), Zurich (Switzerland) and Langenau (West Germany). As a raw water source, the North Saskatchewan River compares favorably with the raw water sources at the locations chosen for comparisons of treatment practice (Foley, Huck, Snoeyink reports, Volume 2). All sources compared favourably with the Canadian Drinking Water Guidelines.[1] The raw water sources chosen for comparison ranged from fair to very good. The North Saskatchewan River rates as a good overall raw water source on a comparative basis.

5.3 Assessment of Any Trends

Reports reviewing historical data, in some cases back to 1963, on the North Saskatchewn River and monitoring data from four locations (Edmonton, Devon, Rocky Mountain House and Whirlpool Point) were reviewed. This review provided no evidence of any trend towards deteriorating water quality upstream of Edmonton. Significant changes in downstream water quality, most of them improvements, can be documented, but that issue is outside the Terms of Reference (Appendix A) for this study.

5.4 Overall Assessment

An assessment of a raw water source should not be based solely on monitoring data. Limitations to the frequency and range of monitoring which can be done prevents such data from telling the entire story.

The presence of upstream discharge sources is an important factor in judging a raw water source. There are only 3 communities, Devon, Drayton Valley and Rocky Mountain House discharging biologically treated sewage directly to the North Saskatchewan River (Figure 1). Each community is less than 5000 population and they are located at distances of 50, 200 and 300 km upstream from Edmonton. Devon and Drayton Valley also disinfect their sewage effluent by chlorination.

The only direct industrial discharge to the North Saskatchewan River upstream of Edmonton is return cooling water from the TransAlta Utilities Sundance coal-fired power plant on Lake Wabamun. This discharge is regulated for pH, suspended matter, total iron and oil and grease. The latter is restricted to 5 mg/L by the Clean Water Act License.

In addition to these direct discharges, there are 14 villages or hamlets which discharge their sewage lagoons, once or twice per year, into streams which flow into the North Saskatchewan River. Finally, the Transalta Utilities Wabamun power plant discharges wastewater from its ash lagoon to Lake Wabamun which eventually drains to the North Saskatchewan River via Wabamun Creek.

Considering the few known point source discharges upstream from Edmonton, the North Saskatchewan River basin would rate as essentially undeveloped in comparison with most other river basin sources providing water to 700,000 consumers.

Spill potential is another factor in assessing a raw water source. In the absence of hard information, spill potential can be roughly correlated with the level of human activity in the river basin. Many spills arise from transportation activities and accidents. There are only six highway river crossings mapped upstream of Edmonton. Of these only four are primary highway crossings (Devon, Drayton Valley, and two at Rocky Mountain House).

There are several pipeline crossings upstream from Edmonton, with most being clustered around Edmonton, Devon and Drayton Valley. These pipelines and other activities involving fuel have been the source of several hydrocarbon spills on the river over the past 30 years. However, these usually result from accidents which will lead to notification of downstream users by emergency response personnel. Such notification has been successful in recent years.

A broad inventory of bulk chemical use in the upstream river basin would be useful for those chemicals which could cause serious water quality problems if spilled. Experience would suggest that the most likely spill scenario would involve hydrocarbon products for which the characteristics and risks are reasonably well known.

The risk of spills within Edmonton affecting the Rossdale water intake, via the storm sewer system, is much more serious than the risk of upstream spills. Drainage from 85 storm outfalls could carry virtually any noxious contaminant discharged, intentionally or otherwise, into the vast storm sewer network. The range of human and industrial activities served by this storm sewer network simply dwarfs the activities occurring upstream of Edmonton.

Experience has indicated the occurrence of several incidents a year involving elevated organic concentrations at the Rossdale intake. These have generally been presumed to arise from storm sewer discharges. The possibility of spills arising from this source, without warning, represents a serious cause for concern. Considering all these factors, the Rossdale water intake must be regarded as highly vulnerable to contamination from spills into the storm sewer system.

5.5 Specific Recommendations

1. Alberta Environment should specifically evaluate the possibility of upstream chemical spills and determine the need for an inventory of bulk chemical usage in the upstream watershed to assist spill monitoring and contingency planning.

2. The City of Edmonton should establish a cooperative network with upstream water users to share information on unusual river water conditions.

3. Alberta Environment should update the Alberta Surface Water Quality Objectives to take account of advances in analytical procedures and water quality knowledge.

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6. ADEQUACY OF QUALITY MONITORING

6.1 Physical and Chemical Quality

Review of the physical and chemical quality monitoring currently being performed on Edmonton water was undertaken primarily by : Dr. Byron Kratochvil, Dr. Ronald T. Coutts and Mr. Albert R. Trussell and Dr. Vernon L.Snoeyink. Dr. Kratochvil assessed the physical and routine chemical analyses and overall quality assurance and quality control. Dr. Coutts assessed trace organics analyses focusing on gas chromatography / mass spectrometry. Mr. Trussell commented upon specialized procedures for taste and odor monitoring and problem solving. Dr. Snoeyink commented on analyses needed for support of process evaluation and improvement.

Dr. Kratochvil reviewed the analyses done and classified them into three groupings: physical and inorganic analyses, chlorine species (including chlorine dioxide) and routine organic analyses. Dr. Kratochvil noted that the first draft of a methods manual was being completed during our review. This is a positive step which is judged to be an essential element of good laboratory practice. Our mandate did not allow an investigation into the history of events in laboratory development. However, an image of a laboratory which has experienced problems but which has recently been making significant strides towards improvement emerged.

Dr. Kratochvil found that the methods in use were generally derived from well recognized sources. Verification of the accuracy of those methods in the hands of laboratory staff was not possible within the scope of this study. Followup procedures to provide that verification are necessary. To provide assurance of accurate, reliable analyses, Dr. Kratochvil stressed the need for blind external testing of all laboratory methods by all analytical staff.

Although the laboratory has recently implemented quality control checks with operators and lab technicians, to be effective, the individual analyst must not be able to know the expected result if performance is to be reliably determined. Because the City contracts out many specialized analyses, they must also be able to verify the quality of the results they obtain from outside labs. In one case, some discrepancies arose between the City results and those contracted out to a commercial lab, specifically on turbidity analysis. Resolution of such discrepancies is important.

Analysis of chlorine dioxide and its inorganic reaction products has been an on-going problem. Since the institution of high-yield chlorine dioxide application in March, 1982, to this spring (1986), the laboratory had been wrestling with analytical methods to support this treatment. The published standard method[2] has been confirmed as incorrect in one aspect and other existing published methods have been found to be too tedious for routine, reliable analysis. Consequently, the laboratory developed a new method which appears to be very promising. With a commitment to further routine verification, the new method should overcome previous monitoring difficulties.

Before these recent developments, the City had been using an inaccurate procedure which may have resulted in considerable unnecessary expense in terms of wasted chemical feed and inadequate control over the treatment process and its byproducts. At the same time as noting this criticism, we should also note that, even with the inaccurate analyses, Edmonton was performing more and better control than many water utilities who have been using chlorine dioxide.[3] However, we should derive limited comfort from the inadequacies of other water utilities in this regard.

Both Dr. Kratochvil and Dr. Coutts reviewed the purge and trap gas chromatographic (GC) method being used for volatile organics monitoring and Dr. Kratochvil reviewed the ultraviolet (UV) absorbance monitoring procedure used. The GC method has been used with an internal standard since March of 1985. That provision allows confirmation that the instrument response is consistent and also allows for a rough semi-quantitative estimation of the amounts of substances responding. However, the laboratory has no means for identifying unknown organic compounds or confirming the presence of known compounds which are detected by GC. Without knowing what is being measured, it is not possible to accurately determine (by calibration with authentic standards) how much is being measured. Thus, although the GC method has proven very useful as an alarm to operational staff when chromatograms indicate the presence of volatile organic compounds, the method, as it now stands, can do little in the way of longer term problem solving or reliable documenting of water supply contamination.

There also needs to be a rationalization of the purpose of the organics monitoring to achieve better monitoring of water quality outside of laboratory hours. This will be considered further in section 6.5.

Although the only method currently available to reliably identify observed GC peaks is gas chromatography / mass spectrometry (GC/MS), Drs. Coutts and Kratochvil agree that the

Edmonton lab should not acquire such instrumentation at this time. This equipment requires a substantial capital and manpower commitment which would be premature given the current state of analytical development existing in the lab. Access to reliable and responsive GC/MS service is, however, essential to achieving the necessary potential of GC for routine monitoring at the treatment plants.

Dr. Kratochvil noted that the UV monitoring is a relatively simple and useful tool for sensing the general presence of organic matter in water. The utility of this method was demonstrated in the discussion of the Spring 1986 rural runoff (Hrudey-1 report, Volume 2). The concept of an upstream UV monitor to provide earlier warning of impending water quality problems will be discussed in section 6.5.

A general assessment of laboratory equipment noted several old instruments which appeared to be well maintained and several newer instruments some of which would qualify as state-of-the-art equipment. Some of the new instruments have not been effectively used because adequate, qualified manpower has been unavailable to implement analytical methods development

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Dr. Kratochvil stresses the need to move towards a computerized laboratory and information management system to achieve efficient and reliable acquisition and processing of routine analytical data. At present, considerable manpower is devoted to recording and transcribing of data which detracts from more productive activities, such as increased quality control and analytical methods development. There is also a morale problem associated with routine recording of numbers which do not change much. Furthermore, the originator of the results may not be able to see them used in a productive manner.

Dr. Coutts reviewed, in some detail, the GC/MS results received by the water labs from contract laboratories. He found many of the results to be poorly documented and some to be wrong in their interpretation. Other results were accurately presented and documented, but the water lab lacked the expertise to interpret the results received. The water lab must have the ability to submit quality control cocktails to insure that results being provided are reliable. In general, a higher level of service will have to be obtained by the City in the future in order to reliably support their organics monitoring program.

Mr. Trussell outlined a number of specific analytical techniques which could be used to better define the nature of the organic contamination and specifically, taste and odour problems. He noted that the continuous odor monitors provided at the plants were a positive step. However, they were located in areas where operators are exposed to a wide range of chemical odours which will make it very difficult for them to sensitively detect odour in the water. Some of these issues will be dealt with further in section 8.5.

Dr. Snoeyink stressed the need to reliably identify the causes of the taste and odour in order to deal with this problem. He also noted the value of determining odour intensity by the threshold odor number (TON), information which was not obtained until the spring 1986 survey.

6.2 Specific Recommendations

1. The Edmonton water lab should implement extractables GC analysis so that non-odorous but otherwise undesirable organics can be identified.

2. The Edmonton water lab should determine the cost-effectiveness of an auto sampler and graphite furnace accessory to their atomic absorption spectrophotometer to allow in-house metals analysis

3. Additional analytical expertise should be acquired to support the methods development function particularly in the organics area and to sustain the advances made in the chlorine dioxide work.

4. Edmonton should institute routine residual aluminum monitoring.

5. The Edmonton water labs should arrange with volunteer office staff at West Chambers to perform periodic threshold odour monitoring on a routine basis to establish odour intensity. This capability needs to be available in advance of major odour events

6. The sources of spring time odour need to be clearly documented to have any chance of reliably eliminating them. The type and intensity of odour in the raw water, that formed during treatment and that formed in the distribution system must be known, otherwise, incorrect responses may be wastefully pursued. This work will have to be backed by substantial analytical support.

7. Alberta Environment should support the City in acquiring research funding to process the organic extracts collected during the spring 1986 monitoring program for high sensitivity GC/MS analysis to document more clearly the nature of raw water chemicals versus oxidation byproducts. Such information will be necessary to effectively plan future treatment responses and depending upon their degree of success, further toxicology testing.

8. Bulk chemicals analysis and materials testing for leachable components have to be a high priority for routine laboratory monitoring.

6.3 Microbiological Quality

Evaluation of the monitoring capability available for microbiological quality of Edmonton water was assessed by four experts. Dr. Richard S.Tobin evaluated monitoring capabilities for bacteriological concerns, Dr. Pierre Payment evaluated monitoring capabilities for waterborne viruses and Dr. Gary S. Logsdon evaluated monitoring for protozoan pathogens, specifically *Giardia*. Dr. Daniel W. Smith also offered recommendations on monitoring of raw water based upon his evaluation of the microbiological condition of the North Saskatchewan River.

Dr. Tobin noted that bacteriological monitoring of Edmonton water is done by the Edmonton water labs and by the Provincial Laboratory of Public Health. The Edmonton water labs perform analyses on raw water, water within the plants, treated water, and water from reservoirs. The Provincial Laboratory of Public Health performs analyses on raw water, treated water from the plants and water samples from the distribution system. For comparison, in the first 3 months of 1986, the Edmonton water labs performed 448 individual bacteriological tests while the Provincial Laboratory of Public Health performed 3367 individual bacteriological tests.

Dr. Tobin found the Edmonton water lab to be minimally capable of conducting useful microbiological analyses. He noted that microbiology work is not done by a specialist, but is just one of the duties of a laboratory technician. While professional supervision is available in the laboratory, routine, direct involvement appears to have been limited. At present, there was no evidence of quality assurance in the analyses being performed. The methods manual, which was only available to Dr. Tobin in draft form, contained a basic error in sampling procedure which has been noted and corrected.

The Edmonton water labs are using the most probable number (MPN) rather than the membrane filtration (MF) procedure for total coliforms analysis. The MPN procedure is more tedious and much less precise than the MF procedure and is only a presumptive test which should be confirmed by further testing. The additional confirmation has not been done by the Edmonton water labs. Standard plate counts using the pour plate method are also performed.

The Provincial Laboratory of Public Health uses the MF procedure for total and fecal coliforms while standard plate counts are done by the pour plate method. Dr. Tobin found the analyses were done by specialist technicians, as a full time activity, under the direct, full time supervision of a professional microbiologist. Dr. Tobin noted that the Provincial Laboratory of Public Health performed daily quality assurance on their analyses. However, he noted that these quality assurance procedures were not currently documented in a methods manual to insure consistent treatment.

Dr. Tobin was concerned that the standard plate count procedure used at both laboratories, despite being a current standard method[2], can under-report the actual number of viable organisms in the sample. Stress placed upon organisms, either by thermal shock during mixing or from the medium being too rich, can cause low counts. The consequences of under-reporting of standard plate counts are significant because these organisms can:

-include some opportunistic pathogens which can infect susceptible persons,
-interfere with the total coliform test when present in large numbers
-contribute to the organic load which coats pipes and increases disinfectant demand
-include coliforms which will cause positive total coliform test results.

There was evidence that under-reporting may be occurring. Dr. Tobin noted that the 1984 Water Treatment Plants Annual Report cited an annual mean value of 6000/L for raw water total coliforms and 4200 for raw water standard plate count. This data does not make sense because the total coliforms should be a subset of the standard plate count. There has also been evidence in the past of high disinfectant demand in the distribution system. Although standard plate counts are not required by the drinking water guidelines[1], this measure provides an important indication of the microbiological activity in the treated water distribution system. The City's initiative in monitoring this parameter is worthwhile and should be continued.

Dr. Smith noted that raw water monitoring for total coliforms, as had been performed until recently, is of little value because of the numerous sources of non-fecal coliforms. These can affect raw water counts without providing any useful information on the possbility of fecal contamination. Daily raw water monitoring for fecal coliforms at both the Rossdale and E.L.Smith plants is necessary for routine documentation of raw water bacterial character.

Dr. Payment reviewed the existing circumstances with regard to viral monitoring and potential contaminaton of water by viruses. He found that only preliminary efforts had been made to perform monitoring for viruses which could affect Edmonton's water supply and there was no available data which warranted evaluation. The practice of viral monitoring is not currently widespread. Dr. Payment notes, however, that the World Health Organization[4] recommends: "where virological facilities can be provided, it is desireable to examine the raw water sources and finished drinking water for the presence of viruses."

Experience elsewhere has shown that contaminated raw water which has been treated to:

-a turbidity of less than 1 NTU

-disinfection with at least 0.5 mg/L of chlorine with a free residual, a contact time of at least 30 minutes and a pH of less than 8.

should be virus-free. Prior to the introduction of chlorine dioxide at Edmonton plants, it is unlikely that the chloramine disinfection and contact time provided at Rossdale (treating a raw water source intermittently contaminated with combined sewage) met this requirement. The turbidity requirement has also been exceeded occasionally. Given limits which may be necessary on the application of chlorine dioxide, the full requirements of the specified level of treatment may not be possible at Rossdale. Hence, the requirements for a virological monitoring program according to the World Health Organization recommendation[3] appear to exist for the Rossdale source, at least until the intake can be relocated. Results from such a monitoring program should provide useful input to the process of developing site specific disinfection criteria (section 7.5)

The E.L. Smith intake should be much less affected by human fecal contamination. Dr. Payment notes that viruses of animal origin do not pose a significant risk to humans. Accordingly, extensive monitoring of the fecal coliform / fecal streptococcus ratio could provide substantial insight into the degree of risk from human virus contamination that may exist at the E.L.Smith intake location.

Dr. Logsdon reviewed the *Giardia* monitoring which has been done and considered the need for further monitoring. The monitoring which was done immediately after the Edmonton *Giardia* outbreak in early 1983 (section 8.4) was done with 7 micron wound element filters. Dr. Logsdon would not expect this technique to be successful for *Giardia* recovery and current practice calls for 1 micron wound element filters. Monitoring at the Rossdale plant the following winter from December 7, 1983 to May 9, 1984 was done with polycarbonate membrane filters which Dr. Logsdon judges to be more reliable. That program did not recover any *Giardia* cysts. Unfortunately, those results cannot tell us anything about conditions at the time of the outbreak.

Results available to Dr. Logsdon from other research has shown that *Giardia* cysts were measured in combined sewer overflows at concentrations of 7 cysts / L with actual concentrations more likely about 70 cysts /L because current sampling methods have low recovery efficiencies. That information means that the possibility of Rossdale raw water being contaminated by *Giardia* cysts certainly cannot be ruled out on the basis of existing monitoring data. Better reassurance can only be provided by a better data base.

Dr. Logsdon notes that *Giardia* monitoring is much more difficult than bacteriological monitoring and that the absence of *Giardia* under limited sampling conditions cannot prove the absence of *Giardia* in the raw water source. He also notes that in-plant monitoring must be based on sampling of individual filters rather than sampling the combined flow from all filters. Presumably, sampling should be directed at the most critical times for particle breakthrough based upon turbidity monitoring. Currently, *Giardia* monitoring is not prevalent in the water treatment industry. Providing assurance of *Giardia* control in Edmonton will require a commitment to undertaking state-of-the-art monitoring procedures.

6.4 Specific Recommendations

1. All coliform analyses done by the Edmonton water labs should be performed by MF procedures using membrane filters known to give high recoveries.

2. All standard plate counts should be performed by MF rather than pour plate methods

3. The microbiological sampling protocol used by Edmonoton waters must be reviewed with all sampling staff to provide clear explanation for the reasoning behind specified procedures.

4. When total coliform are found to appear in treated water and in the distribution system these should occsasionally be identified as to species.

5. The quality assurance and quality control procedures at the Provincial Laboratory of Public Health (PLPH) should be documented in a methods manual. This would form a useful reference for other labs such as the Edmonton water lab.

6. The PLPH should engage in comparative test studies with another major lab in western Canada for mutual verification.

7. Because temperature control of +/- 0.2°C is crucial for the fecal coliforms test, an NBS-certified thermometer should be installed on the incubator and a written log of daily temperature maintained.

8. At least 10% of the samples should be devoted to quality assurance testing and reference procedures noted by Dr. Tobin should be followed for quality assurance and control.

9. The raw water should be monitored daily for fecal coliforms . Furthermore, fecal streptococcus monitoring should be instituted on a regular basis at E.L.Smith to establish more clearly the degree to which fecal contamination can be attributed to animal versus human origin.

10. A limited program of virological monitoring, as outlined by Dr. Payment, should be instituted to define the extent of viral contamination at Rossdale and to provide input to the decision making process concerning interim disinfection practice until the intake can be relocated.

11. The need for extensive viral monitoring at E.L.Smith may be clarified by review of the fecal coliform / fecal streptococci ratios, but some monitoring while the Rossdale program is underway would be prudent.

12. An overall risk assessment to evaluate the likelihood and circumstances associated with *Giardia* contamination of the raw water should be performed. Monitoring for *Giardia* cysts

in raw water at Rossdale should be reinstated on a regular basis to provide input to this risk assessment and to decision making on disinfection requirements. Because the Rossdale intake should be moved to a location which would take water equivalent in quality to E.L. Smith, the emphasis in monitoring should shift to characterizing the likelihood of cysts occurring at the E.L. Smith intake.

6.5 Emergency Warning Capability

At present, the raw water at Edmonton plants is periodically monitored for UV absorbance and volatile organics by gas chromatography. While the GC method has identified contamination and allowed corrective action, the monitoring is not continuous. Likewise, UV absorbance measurements have documented the arrival of river water which is high in dissolved organic carbon, but this monitoring is also not performed on a continuous basis.

Continuous monitoring of raw water for utilities would seem to be a high priority, but very few scientific papers have been published on this topic. Of the parameters which are reported for continuous monitors, only those related to organics are particularly relevant. In this case, automated discrete analyzers for total organic carbon, ultraviolet absorbance (UV) and fluorescence seem to offer the most promise. Given the nature of contamination problems historically experienced at Edmonton plants, a combination of continuous UV and fluorescence monitoring would likely be most cost-effective because of the simplicity of operation and low cost for the necessary instrumentation.

Most oil contamination which would pose a concern to the water intake should have enough aromatic character to fluoresce. This type of detection would be useful at Rossdale where oil contamination from urban runoff has been a re-occurring problem.

Continuous UV absorbance monitoring would be most useful at an upstream location, such as Devon or Genessee, to give advance warning of the annnual arrival of peak dissolved organic carbon levels. The past two years, this peak has coincided with high chlorine dioxide demand and the well known odour incidents. Advance warning of the peak dissolved organic carbon would allow the plants to adjust treatment process conditions and perhaps allow better response to these circumstances.

Fish biomonitors have been actively considered by the Edmonton plants. Calgary is currently evaluating a fish device at its Glenmore plant. Unfortunately, these devices have not yet proven their capability to respond to a wide enough range of contaminants at low enough concentrations to justify their utility for overall system protection.

6.6 Specific Recommendations

1.Edmonton should seek to install a continuous flow UV monitor initally at the Devon water treatment plant and, if found necessary, at the Genessee power plant to provide advanced warning of the spring dissolved organic carbon peak

2. Edmonton should perform a trial with continuous fluorescence monitoring at Rossdale to determine if it is sufficiently sensitive to provide warning of hydrocarbon contamination. This can be done by comparison with existing periodic GC monitoring.

3. Edmonton should evaluate the utility of purgeable organic carbon as a semi-continous monitoring parameter by performing some cooperative analyses with the Department of Civil Engineering at the University of Alberta.

4. Edmonton should confer with Calgary over their experience with a continuous fish monitor to determine if there would be any value to that investment in Edmonton.

6.7 Overall Assessment

Review of the monitoring being performed indicated several areas which need improvement. The major concerns involved quality assurance and quality control. Clearly, data generated on drinking water quality must be reliable to have any value and positive assessment of the capability of the laboratory cannot be given until adequate quality assurance is in place. There is cause to believe that the necessary changes are recognized and will be implemented, considering the advances apparently made in lab operations in recent years.

The need for in-house laboratory capability for the City must be considered in light of the extensive monitoring performed by Alberta Environment as part of the Treated Water Surveys. Because the City of Edmonton has the responsibility of treating water to Clean Water Act license conditions while satisfying its paying customers, it must maintain a realistic capability to judge performance. Total, or even major, reliance on outside agencies to determine performance would undermine the pool of in-house expertise and knowledge necessary to run a major water utility in an efficient, cost-effective manner. The monitoring provided by Alberta Environment should be viewed as both an audit and a general support function.

7. CURRENT TREATMENT PRACTICE

7.1 Adequacy of License Requirements

The adequacy of the Edmonton water facilities Licenses to Operate (under the Clean Water Act) was assessed and compared with practice in Ontario and Quebec by Mr. Paul D. Foley. He found that Alberta practice was at least equivalent to Quebec where drinking water standards are incorporated into their legislation. In Alberta, the Canadian Drinking Water Guidelines[1], except as modified by Alberta Environment, are incorporated as license conditions. Consequently, they are enforceable under the legislation. Based on current regulations, Alberta Environment can only modify license values from the Canadian Drinking Water Guidelines[1] for non-health related parameters.

Ontario is more informal in its reporting requirements. The Ministry of Environment has been providing analytical services for most water treatment plants, except for the very large municipalities. Alberta also provides an analytical service for meeting regulatory requirements under the Clean Water Act. In the case of Edmonton, this service is covered by the Treated Water Surveys. Overall, Mr Foley rated the Alberta regulatory practice at the forefront of practice, as he understands it, in Canada. The only specific deficiency noted in the existing license, was a need for requiring a formalized quality assurance / quality control program.

Some related issues have arisen during the course of this study which could be considered in this context. Firstly, it is not entirely clear what the respective roles of the City and province are with respect to raw water quality monitoring. There appears to be scope for more cooperative exchange of information and data gathering on the North Saskatchewan River.

Secondly, it seems that there should be scope for the City to have analytical methods development support from Alberta Environment and the Alberta Environmental Center (AEC). Presumably, major problems identified by Edmonton could and should be included in the research priorities of Alberta Environment and the AEC. Likewise, the City could have valuable inputs to the planning of environmental studies which may include the upstream water shed. Seeking input to such exercises would be in the best interests of the City with regard to understanding the quality of the raw water source.

Thirdly, there appears to be scope for actively involving major cities in river basin planning exercises in order to allow them to voice their interests in watershed protection.

7.2 Comparison with Canadian Practice

Mr. Foley visited the Rossdale and E.L. Smith plants (Figures 6, 7 and 8) in December, 1985 and directed his evaluation of Edmonton practice to comparsions with practice in Ontario and Quebec. He found that, in general, the unit processes used in Edmonton are similar to those used elsewhere with only minor differences in specifications. The values for some operating parameters, under maximum water demand conditions, were judged to be at the maximum desirable level.

Filtration performance, as indicated by an average of 0.5 NTU in 1984 was noted to be behind typical Ontario performance of 0.3 NTU. Mr. Foley endorsed a goal of 0.1 NTU as a worthwhile objective with many ancillary benefits. Methods for achieving that goal would involve more bench scale testing with jar tests to determine optimum treatment conditions. There must be a recognition that the existing plant and raw water source my not allow the 0.1 NTU goal to be routinely achieved. However, the value of striving for that target is a theme which will re-occur throughout the expert reports on treatment.

Several Ontario plants were considered for comparison with Edmonton. Because 80% of Ontario's drinking water comes from the Great Lakes, most of their treatment problems differ from those in Edmonton. Specifically, most plants encounter taste and odor problems but these arise from algae and are successfully controlled with chlorine. Comparison with Ottawa was informative because they use a river source and they found the need to relocate one of their intakes to avoid bacterial contamination (also discussed by Dr. Smith, Volume 2)

Plants in Quebec use river sources more commonly and they have experienced many taste and odor problems. In particular, Chambly and St. Jerome have been using chlorine dioxide and they have experienced swampy odors at high chlorine dioxide dosages. Unfortunately, these problems have not been adequately documented and they are not useful for solving Edmonton problems. Ozone is widely used at a number of plants in Quebec for taste and odor control and as

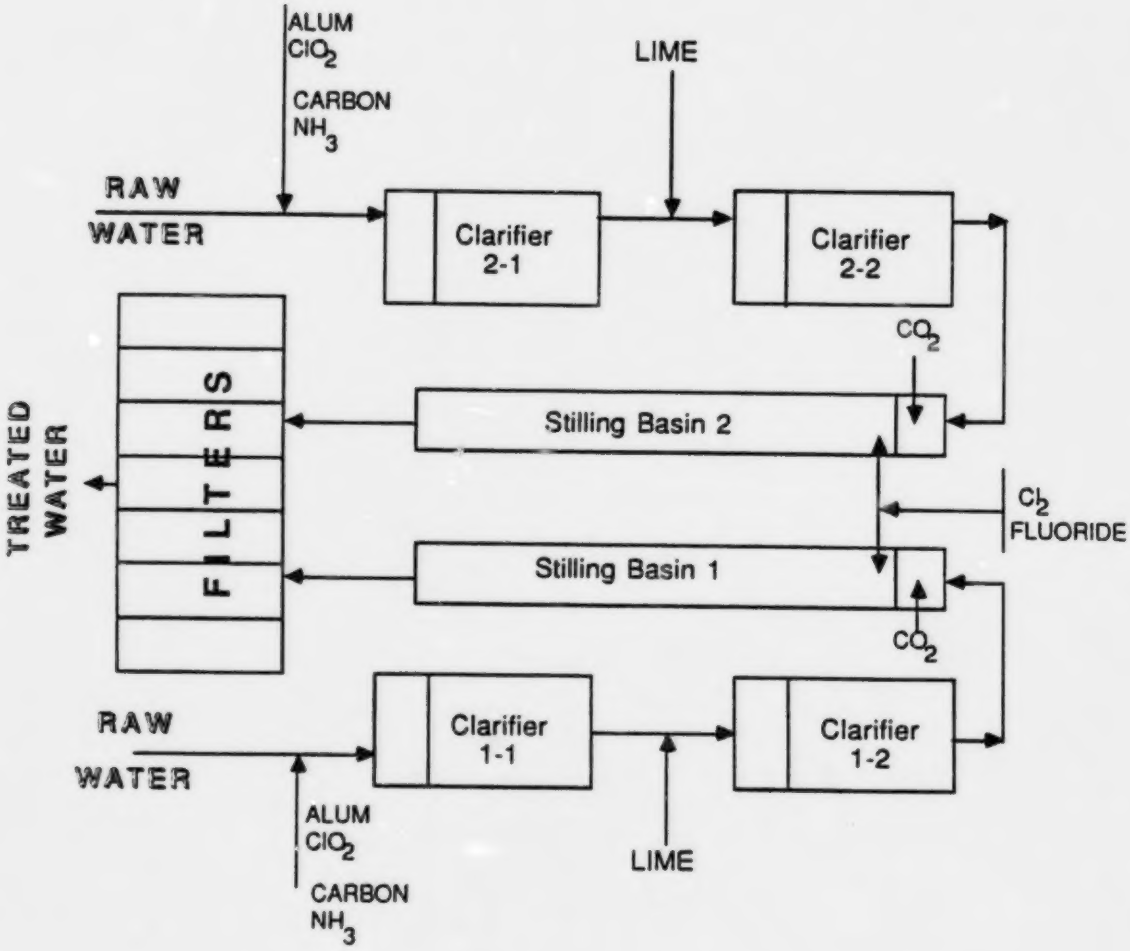

Figure 6. Process Schematic for Rossdale Plants 1 & 2.

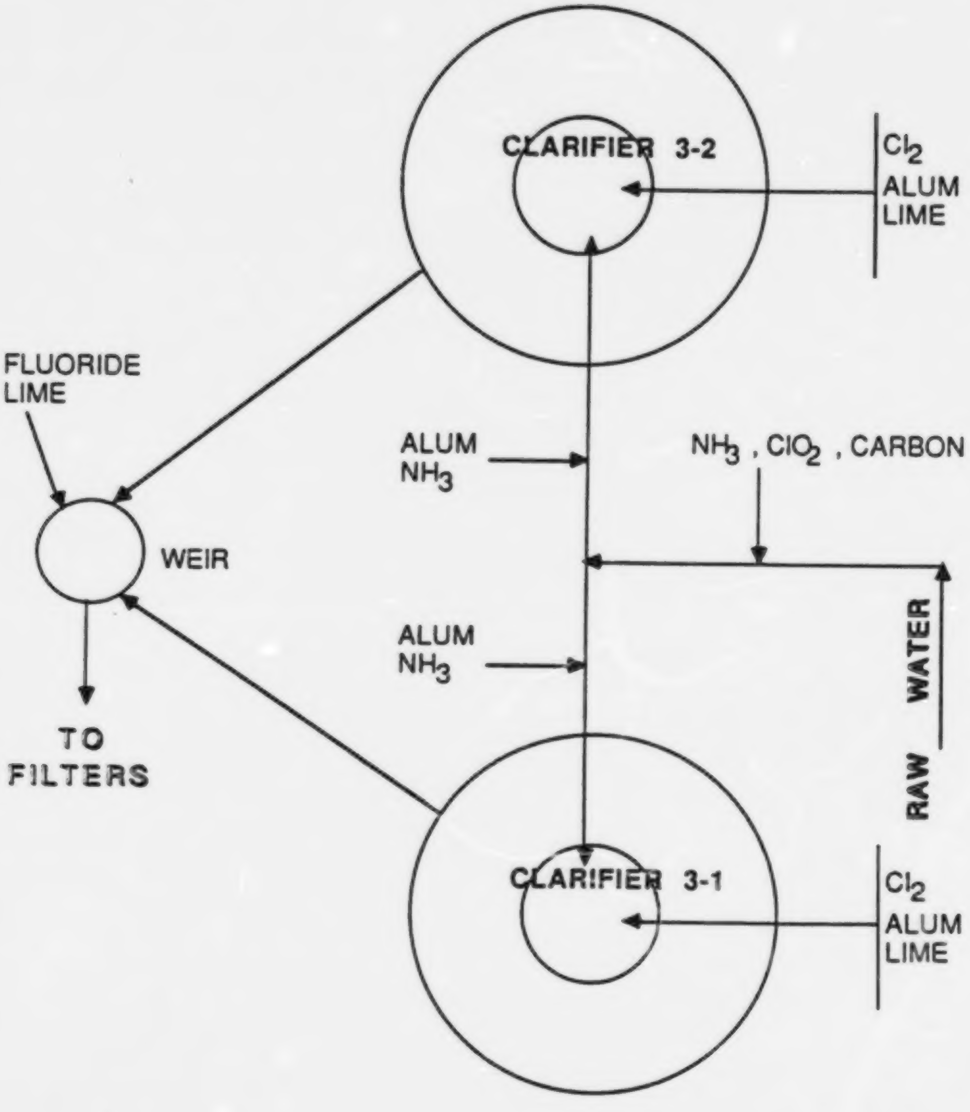

Figure 7. Process Schematic for Rossdale Plant 3.

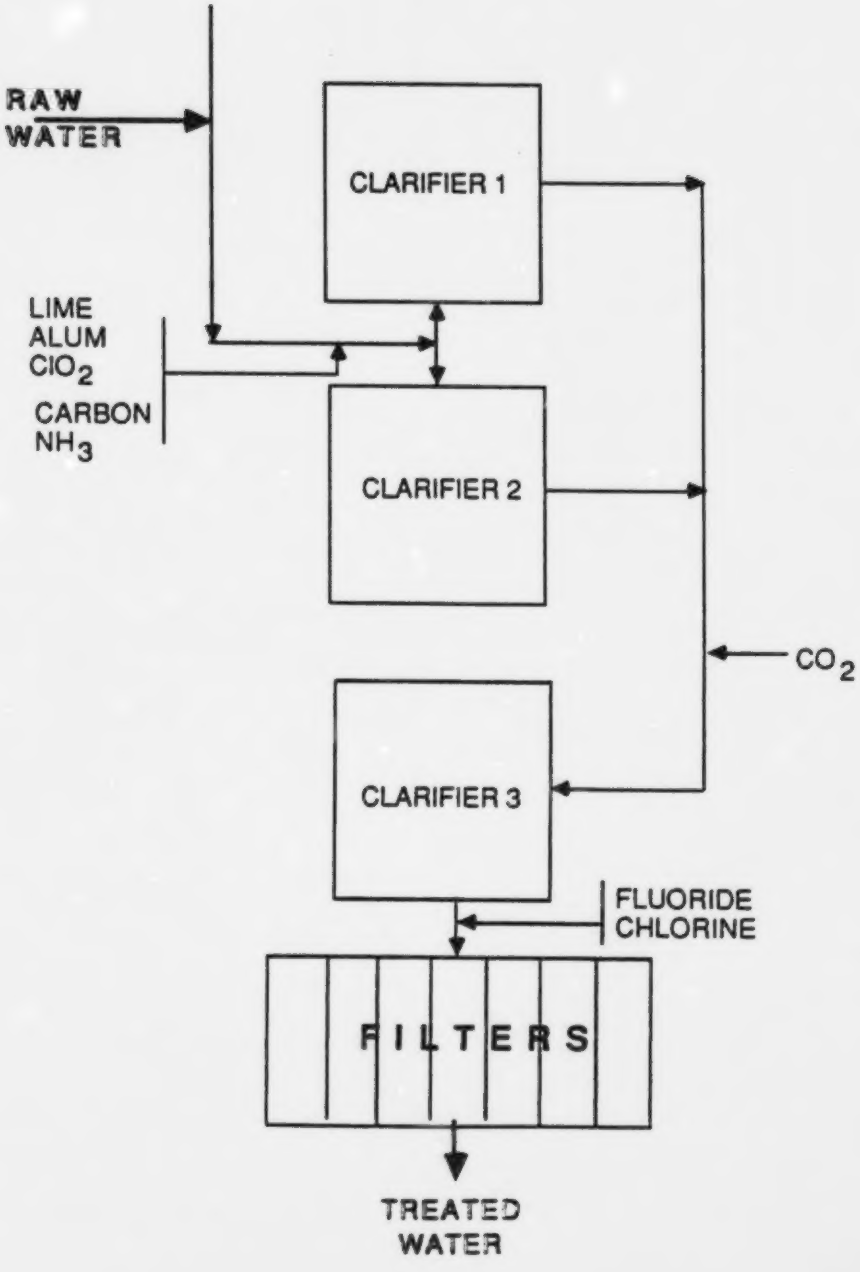

Figure 8. Process Schematic for E.L. Smith (Plant 4).

a primary disinfectant. Quebec experience should prove valuable to any future evaluation of ozone in Edmonton.

Overall, Edmonton would appear to be no more prone to problems than most other water supplies in Ontario and Quebec. Each community is likely to experience somewhat unique problems which necessitates having a trouble-shooting team to identify and solve problems as they arise. This team concept will be developed further with discsussion of subsequent expert reports.

7.3 Comparison with U.S. Practice

Dr. Vernon. L. Snoeyink evaluated water treatment practice in Edmonton and compared it with practice in the United States. Dr. Snoeyink visited the Rossdale and E.L. Smith plants in January, 1986. He observed that the E.L. Smith plant is a modern water treatment plant which generally appears to be well run. The older Rossdale plant was found to be in need of the upgrading which was underway. Overall, Dr. Snoeyink found many problems were identified by plant staff, but relatively little hard information had been collected to document specific causes of problems and provide a rational basis for developing solutions.

The obvious problems with taste and odor, which will be discussed in section 8.5, were judged to be only symptomatic of a broader need to be able to: rigorously study problems, develop a clear understanding of the factors involved, and effectively implement solutions through orderly process adjustment or development.

Dr. Snoeyink proposed several process alternatives which merit consideration. In all cases, considerable characterization work is necessary before proceeding with implementation of these suggestions. Thereafter, process improvement by a specialist team consisting of at least an engineer, a chemist and a technician will be necessary to insure effective implementation. Dr. Logsdon also elaborated on this concept in section 7.5.

Some specific process problems were noted which require early attention. Addition of ammonia, alum, powdered activated carbon and chlorine dioxide all at one location is counter-productive (Mr. Foley and Dr. Huck shared concerns over this practice). Since, March the point of chlorine dioxide addition has been moved to between clarification and softening at E.L.Smith and the chlorine dioxide addition point was being moved at Rossdale, so this concern has been partially addressed. There is still a concern that the powdered activated carbon will be less effective without having some raw water contact time before alum addition.

Dr. Snoeyink noted high solids carryover from the softening stages to the filters. He agrees with all the other process experts about the need to reduce filtered water turbidity and sees reduction of the solids loading from softening as an important starting point. A need for more jar testing to achieve such improvements was outlined.

Dr. Snoeyink also expressed concern over the possiblity of high dissolved aluminum arising from the relatively high pH employed during winter coagulation at E.L.Smith. Monitoring of residual aluminum and process pH control to reduce soluble aluminum to below 0.2 mg/L, with a target of 0.05 mg/L, are necessary.

Both Dr. Snoeyink and Mr. Trussell expressed concern over the selection and purchasing of powdered activated carbon (PAC). In the past, PAC has been selected on the basis of lowest bidder without regard to performance on the chemicals intended for removal. Because carbons can vary widely in performance, this is an important issue. Edmonton Water and Sanitation has sponsored a research program this year at the University of Alberta to tackle this problem.

Mr. Trussell also expressed concerns over the lack of monitoring and control of ammonia nitrogen levels. Without maintaining a proper balance between ammonia added initially and chlorine added later, with taking account of the ammonia content of the raw water, a variety of undesirable reactions can occur, some of which will contribute to odour.

Two U.S. water treatment utilities, Hays Mine Station in Pittsburgh and St Louis County, Missouri were selected by Dr. Snoeyink for comparison with Edmonton. Both face poorer raw water quality than Edmonton, but there were analogies to the situation existing at Rossdale. Hays Mine Station is downstream from many industrial discharges containing benzene and phenol. Raw water benzene levels up to 80 ug/L (ppb) have been experienced and frequent raw water monitoring is necessary. Threshold odor numbers of 60 occur in the river with typical values in the 5 to 9 range. The plant uses powdered activated carbon and granular activated carbon media in their filters. Treated water is produced without odor and only detectable quantities of benzene.

Also the finished water turbidity which averages 0.15 to 0.2 NTU (although a maximum of 4.5 was reported) is significant.

St. Louis County uses very turbid raw water from two rivers but produces water with an average finished turbidity of 0.15 NTU (maximum not reported). They have found that complaints about odour in the distribution system will arise when treated water turbidity rises much above 0.2 NTU. This utility, which averages 540 ML production (compared with 300 ML for Edmonton), receives about 350 taste and odour complaints a year (compared with 100-200 /yr in Edmonton in recent years).

Overall, Dr. Snoeyink did not identify any gross deficiencies with water treatment practice in Edmonton compared with large utilities in the United States. There were, however, many areas identified where substantial specific changes should be made and substantial improvements can be pursued. Acting upon these recommendations will require a dedicated process improvement team which is free from daily operational responsibilities.

7.4 Comparison with European Practice

Dr. Peter Huck reviewed treatment practice at three advanced European facilities to determine any applications which may be useful for Edmonton. On reviewing Edmonton's practice, Dr. Huck shared the concerns about simultaneous addition of several chemicals to the raw water, high turbidity applied to the filters and the need for process optimization, particularly for organics removal. He selected facilities at Zurich, Switzerland, Langenau, West Germany and Mery-Sur-Oise, France, for detailed evaluation.

Zurich provides a case where the raw water source is better than that available to Edmonton. However, this facility experienced a taste episode with phenols approximately 15 years ago which caused them to explore alternate and advance water treatment. The main features of interest to Edmonton is their use of a combination of oxidant chemicals and their demonstrated success with a commitment to detailed process development and improvement.

Langenau provides a case where the raw water source is somewhat similar to the North Saskatchewan River. This facility demonstrates remarkable turbidity removal and provides another testimonial to the achievements of a commitment to process development and improvement.

Finally, Mery-Sur-Oise is a case where the raw water source is substantially poorer than the North Saskatchewan River. Notwithstanding, their treated water quality is excellent. Once again, process evaluation and development have been used to optimize system performance to deal with raw water quality challenges. Notable features here include their use of aluminum polychloride as a coagulant and their strict separation of unit processes.

Overall, these evaluations illustrate a range of treatment options coinciding with raw water ranging from poor to excellent. A preference for ozone as primary disinfectant and chlorine or chlorine dioxide as protection for the distribution system is apparent. Processes are tending towards maximizing the biological stability of the treated water using ozone and biological activated carbon to minimize the demand for disinfection chemicals. By so doing, the problems which may be associated with the formation of disinfectant byproducts are minimized. Likewise, optimization of turbidity removal maximizes protection against viruses and protozoan cysts while minimizing disinfectant chemical demand.

We were also fortunate to have input from Professor H. Bernhardt from the Federal Republic of Germany concerning the question of removal processes for natural organics. He has proposed a process scheme which could be evaluated at E.L. Smith to deal with the high dissolved organic carbon experienced at peak spring runoff. Professor Bernhardt also emphasized the need to move the Rossdale intake upstream of the influence of the storm sewers because he notes from his extensive experience that treatment processes are not as reliable as good source selection and protection.

7.5 Overall Assessment

Several recurring themes arise from the views of the various treatment experts who visited the plants. These are well expressed in Dr. Logsdon's report which included a detailed assessment of the capability of the Edmonton plants to remove *Giardia*. Dr. Logsdon questions whether enough use is being made of available or readily obtainable process control information. Intermediate process operating targets should be set and used as indicators of when intervention and operating changes are necessary to deal effectively with variable raw water quality.

The treatment experts generally agree that there is considerable scope for improving existing facility performance before investing in substantial changes to the treatment processes available. The implementation of orderly process evaluation studies will be the key to cost effective improvements. Beyond the bench scale evaluations, which are critical to initiating process improvements, there would be considerable value in having a small scale pilot facility to prove out the performance of process alternatives. Without such pilot facilities, the water treatment staff have to experiment on the consumer by implementing processes full scale. That type of process development is undesirable and, in the long term, rarely cost-effective.

Dr. Logsdon also notes that availability of a pilot facility will prove valuable for operator training. By being able to directly demonstrate what effects different operating procedures can have on treated water quality, without risking the water under full scale production, important lessons can be taught. Judicious use of educational and professional development opportunities for career employees will also pay dividends in terms of capable and cost effective operation. Maintaining a high level of in-house expertise in water treatment and quality assessment is very important to successful operations.

Concerns expressed by several experts about the adequacy of disinfection require specific attention. Because no disinfectant is free of health concerns there is a need to avoid overzealous use of disinfectants. On the other hand, the possbility of allowing transmission of waterborne disease because of inadequate disinfection is unacceptable. While considerable guidance on disinfection practice can be gained from research and experience elsewhere, Edmonton should undertake to evaluate the critical controlling factors governing disinfection needs under Edmonton conditions and develop a site specific disinfection approach. Factors such as water temperature, pH and potential disinfectant demand should be incorporated into disinfection trials with the most resistant plausible pathogen (likely *Giardia*). Results should be used to derive the concentration and contact time requirements which must be provided for the available disinfectant options. Based on these results, an operational code for disinfectant dosage and reservoir operation to provide assured contact time can be developed.

Finally, most experts who visited the plants expressed, verbally, the impression that operational staff were keenly interested in doing a good job and could be expected to respond positively to constructive suggestions for improved operating procedures.

7.6 Specific Recommendations

1. Several specific process options were proposed by Prof.H. Bernhardt, P.D. Foley, P.M.Huck, G.S. Logsdon, V.L.Snoeyink and A.R. Trussell and these should be carefully considered for followup.

2. An enhanced and formalized quality assurance and quality control program should be instituted in the plant to verify flow meters, head loss gauges, chemical feeders and similar equipment for the accuracy of their performance. Likewise, residence times in treatment units should be periodically verified.

3. Means for discharging inadequate quality water which is in the process at Rossdale plants 1 and 2 should be available without having to drain the plant.

4. Efforts towards reducing dependence upon heated water at Rossdale should continue. This an example of substantial savings which can arise from re-evaluating past practice.

5. The point of chlorine dioxide addition must be downstream of alum coagulation and clarification. This is necessary to obtain the benefit of removing raw water organic load by clarification before undesirable reactions with the oxidant can occur.

6. Chlorine dioxide should not be added simultaneously with powdered activated carbon. The carbon will exert a demand upon the chlorine dioxide with concurrent wastage of the feed chemical (sodium chlorite).

7. The point of chlorine dioxide addition must be provided with rapid mixing. Otherwise, very high concentrations of chlorine dioxide can be sustained in contact with organic matter in the

water. This could result in formation of many unknown byproducts which would not be formed at fully mixed dose levels.

8.The need for continued practice of chorine dioxide disinfection at E.L.Smith should be demonstrated.

9. Chorine dioxide use carries a responsibility to maintain careful control over generator efficiency, dosage levels and measured residuals. That responsibility requires a high level of analytical competence which can only be assured by direction from highly trained analysts.

10. Alberta Environment through the Alberta Environmental Center should assist Edmonton in developing further analytical improvements for chlorine dioxide and chlorite. Such methods development should also result in inclusion of chlorite and chlorine dioxide in the Alberta Environment Treated Water Surveys for communities using chlorine dioxide.

11. Use of chlorine dioxide should continue to be restricted to dosages which will produce a maximum residual of 0.5 mg/L of the combination of chlorine dioxide plus chlorite. This, however, must be viewed as an interim measure until the Rossdale intake can be moved and/or further guidance can be obtained for a suitable regulatory level. The level must also be viewed as a true maximum and not an average target. Review of records of past dosage levels suggests that a target of less than 0.3 mg/L is probably realistic for normal operating conditions.

12. Chlorine dioxide application should be cross-checked by mass balance controls on sodium chlorite consumption, much as are currently used for fluoride control. Operators can assure the maximum possible dose of chlorine dioxide is limited to set levels by checking the mass quantities of raw material fed to the generators.

13. Because disinfection practice is central to so many issues, the City must accurately verify the contact times in treated water reservoirs at the plants.

14. The city should implement their own site-specific, performance-oriented specifications for bulk chemical purchases, notably powdered activated carbon, to ensure the most cost-effective treatment will be achieved.

15. The city should adopt a long term goal for its process improvement team to maximize the biological stability of treated water. By minimizing the presence of available organic material which can promote downstream biological activity in the distribution system, the disinfectant

demand in the distribution system can be minimized. Consequently, disinfectant dose can be minimized without compromising protection against pathogens.

16. The process improvement team should consider very carefully the value of short-term flow-through raw water storage with the option of open-air pretreatment for organics during the short term peaks in North Saskatchewan River dissolved organic carbon.

8. TREATED WATER QUALITY

8.1 Compliance with License Requirements

Performance of the Edmonton Water Treatment Plants, based upon review of their Annual and Monthly reports, was assessed in the Hrudey-1 report (Volume 2) and can be categorized into general issues and specific cases of license violations.

Dealing first with license violations based upon Edmonton data, there was a distinct decrease in the number, significance and degree of license violations over the review period (1980 to May 1986). Only temperature violations, which are largely beyond the control of plant personnel, and odor complaints escaped this trend. Occasional very high metals violations which occurred in the early 1980's have not been noted in recent years. Frequent fluoride and occasional pH, color and turbidity excursions have been largely eliminated. Marginal phenols violations still occur but with less frequency and severity than were evident in the early 1980's. Phenols violations have not been correlated with taste and odor complaints. Problems with maintaining licensed levels of chlorine residual in distribution reservoirs also appear to have been overcome.

The current practice of trying to meet objective levels which are below the license limits for several parameters is commendable and is likely beneficial in assuring good license compliance.

In conclusion, the current level of compliance with the Clean Water Act licenses, based upon Edmonton's records, is excellent, particularly in contrast to the blemished record only a few years ago.

A review of Edmonton's performance based upon independent, provincial data was also performed in the Hrudey-1 report (Volume 2). Alberta Environment has conducted treated waters surveys on Alberta's major communities since 1978 and uses this data to insure license compliance. The scope of these studies has steadily increased. The 1984 and 1985 surveys involved samples collected once per month at taps in the distribution system for each of the Smith and Rossdale plants. A total of 204 parameters were analyzed for each sample.

Results for Edmonton Rossdale and Edmonton E.L.Smith for 1984 and 1985 were mainly evaluated for health-related parameters from the Canadian Drinking Water Guidelines.[1] Review of Rossdale results indicates that most parameters are present at insignificant levels relative to the guidelines. The only exception was iron which peaked at about 80% of the license limit in

July, 1984. The iron limit is specified for aesthetic rather than health reasons, but should not even pose an aesthetic problem for an individual sample at that level. Other metals with measurable values peaked at the following fractions of the license limit: 40% (cadmium), 20% (chromium) and 10% (copper).

Review of E.L.Smith results indicates that most parameters are also present at levels insignificant relative to the guidelines. The maximum value was a single observation for cadmium at 60% of the license limit. Chromium and mercury peaked at 20% and copper at 10% of their license limits.

These results are important in three ways.

1. They provide important, independent corroboration that Edmonton's water is meeting health-related guideline values.
2. Samples are collected more frequently (once per month) than City sampling for parameters such as the metals.
3. Samples are collected in the distribution system and consequently they give information on water quality in the distribution system.

The results provided by the Alberta Treated Water Surveys confirm the conclusions that the quality of Edmonton drinking water for health-related license limits is excellent. With the exception of taste and odor, which is not part of the survey, results are also good for aesthetic parameters.

8.2 Comparison with Treated Water Quality Elsewhere

Treated water quality data from both of Edmonton's water treatment facilities, Rossdale and E.L. Smith were compared in the Hrudey-1 report (Volume 2) to water quality data summaries obtained for: Calgary (Alberta Treated Water Survey Data), Ottawa and Brantford, Ontario; St. Louis County, Missouri; Langenau, West Germany; and Zurich, Switzerland. Data was available for a limited set of health-related parameters derived from the Canadian Drinking Water Guidelines.[1]

A comparison with limited trace organic contaminant data available from other cities was also performed in the Hrudey-1 report (Volume 2). This review indicated that the data base available for Edmonton from the Alberta Treated Water Surveys is more comprehensive than any which could be acquired for other cities. Comparing the limited data which was acquired from elsewhere indicated that trace organic substances do not appear to normally occur with any greater frequency or concentration than elsewhere. This generalization cannot be extended to periods with problem conditions, such as spring runoff, because equivalent data from other locations was not available.This overview assessment indicated that for the parameters for which data is available, Edmonton water compared favorably to all these other locations.

8.3 Health Effects of Chemical Substances

8.3.1 Trace Organic Contaminants Identified in Edmonton Water

Data from special surveys by the City of Edmonton and Alberta Environment, the Alberta Treated Water Surveys and the 1986 City of Edmonton monitoring program were reviewed for information on trace organic substances in Edmonton water. The data available by early March 1986 was referred to the Canadian Centre for Toxicology in Guelph for individual assessments of those compounds identified with confidence. The health significance of these compounds is reviewed in detail in the Canadian Centre for Toxicology report (Volume 2).

Of the compounds identified, four were considered carcinogens: benzene, chlorobenzene, methylene chloride and chloroform. Of these, all but chloroform represented less than 1 in a million lifetime risk of cancer via water consumption, the risk level generally accepted as insignificant.

For chloroform, the average concentration in Edmonton water for 1984 and 1985 was 2.4 ug/L(ppb) which corresponds to a level 15 times lower than the guideline for chloroform proposed by the World Health Organization[4] and 40 times lower than the U.S. Environmental Protection Agency standard[5] for trihalomethanes (of which chloroform is usually the major component). The maximum observed concentration was 11 ug/L(ppb) which is 32 times lower than the maximum acceptable concentration for trihalomethanes specified by the Canadian Drinking Water Guidelines.[1]

Several of the compounds could not be adequately assessed because of a lack of specific toxicological data. Where possible, analogies or other deductions were made to comment on toxicological significance. This situation of uncertainty is addressed by Dr. Bull in his overall assessment of the health risk posed by compounds found in Edmonton drinking water (Bull report, Volume 2; section 9 this report).The limitations of drawing inferences about health risk from assessment of individual chemicals is also addressed.

There appears to be some possibility that some of the rarer compounds found are oxidation byproducts of chlorine dioxide. Steps to minimize formation of such compounds are discussed in section 10.3.

8.3.2 Health Issues Raised by Public Input

Written submissions were received from citizens, as outlined in section 3. These submissions were reviewed to insure that the study would address the concerns of citizens who had taken the trouble to write. In a few cases, the specific concerns expressed were not being explicitly studied by any of the experts retained. Consequently, Dr. Robert E. Rogers of Toxcon Consulting Ltd, a local toxicologist, was retained to address these specific concerns. Constraints of time and budget limited the scope of Dr. Rogers' inquiry to a brief review of scientific literature on the various questions and a limited evaluation of Edmonton water quality data with regard to the topics raised.

Five issues were referred to Dr. Rogers for literature assessment. These were:

1. the relationship of water hardness to heart disease
2. the relation of waterborne sodium to heart disease
3. risks to a developing fetus from drinking Edmonton water
4. potential adverse health effects of fluoridation of water
5. relationship of drinking Edmonton water to Lou Gehrig's disease.

Edmonton currently performs limited lime softening of North Saskatchewan River water, to reduce hardness by 30 to 50 mg/L to levels of about 125 mg/L(as $CaCO_3$), on average. These levels correspond to water which would be described as moderately hard, although at the lower end of that designation. Dr. Rogers investigated the case for an association between water hardness and reduced heart disease. He concluded that no case was proven from existing studies. Notwithstanding the lack of a proven health benefit to hard water, there do not appear to be compelling reasons for any further softening of Edmonton water which would justify the additional costs involved.

The possible association between sodium intake and heart disease was evaluated for the importance of sodium levels in Edmonton drinking water. The latter are very low, typically around 4 mg/L. With such low values, sodium intake from drinking Edmonton water will be a small fraction of daily intake from all sources, including food. The link between sodium uptake from water and heart disease is not established. In the specific case of Edmonton, the very low sodium levels do not appear to pose any risk . Of course, anyone on a strict sodium diet should consult their physician about the need to restrict even the very limited sodium uptake possible from drinking Edmonton water .

Dr. Rogers reviewed the literature for evidence of drinking water effects on the developing fetus. He found very few epidemiological studies considering the role of drinking water on fetal development. Most available information was directed to the possible effects of specific chemical substances. Approaching the problem from this perspective, Edmonton water was well below maximum acceptable concentrations[1] for substances believed to have an effect on fetal development. Likewise, intake from drinking water was found to be low relative to intake from other sources such as food. Based on the substances which could be evaluated by this approach, the risks would appear to be low.

Because many of the trace organics which occurred during the spring odor event do not have an adequate toxicological data base, very little can be inferred about the effects of such substances on the developing fetus. Some comfort can be drawn for those substances which are naturally present as components of food. As with any other consumer, the concerned, expectant mother should not feel compelled to drink water if she finds it to be offensive in taste or odour. Otherwise, we have more assurance of safety for Edmonton drinking water than can be provided for alternative water sources, such as bottled water, because there is no routine quality monitoring required for such alternate sources.

Dr. Rogers reviewed the literature on fluoridation of drinking water and found there was a lingering controversy over potential health effects from fluoride. Dr. Bull has also commented on this issue and noted that scientific findings seem overshadowed by social debate on the subject.

While Dr. Rogers reports limited evidence to implicate fluoride for mutagenic effects in cell cultures, Dr. Bull notes that such screening techniques cannot take precedence over epidemiological data which is certainly not lacking in this case. Considerable attention has been directed to the possibility of fluoride being carcinogenic and epidemiological data provide no evidence to support that prospect.

In considering fluoride in Edmonton drinking water, it is worth noting that natural levels from the river comprise 10 to 15 % of the levels in the final drinking water. Hence, even if Edmonton were not intentionally fluoridating the water, exposure would not be negligible as some might assume. I am inclined to accept Dr. Bull's view that there is not convincing evidence of adverse health effects associated with the level of fluoridation used by Edmonton, provided dosage is carefully controlled and monitored. I also accept the view that this position should be regularly reconsidered as new health information arises. The previous commitment to adopt this practice should not prevent our health authorities from critically re-assessing its benefits, costs and potential health effects if convincing new evidence should arise. Because there is substantial evidence of beneficial dental health improvement for Edmonton's youth since 1967 when fluoridation was adopted, and the annual cost is small, the value of fluoridation in Edmonton seems apparent.

Dr. Rogers considered the possibility of a relationship of the rare, Lou Gehrig's disease, amyotrophic lateral schlerosis (ALS) to drinking Edmonton water. He found that causation has not been determined for this illness and there is no basis to evaluate a relationship between Edmonton drinking water and the disease.

In addition to the specific health issues raised, there were ten letters received that described health effects (other than *Giardia* infection) which the respondents attributed directly to drinking Edmonton water. Dr. Rogers was able to locate eight of these individuals for an interview. He found that most of the respondents regarded themselves as normally healthy. Effects of other illnesses did not seem to be a common factor in their complaints. There were six of the eight who experienced allergies which may suggest a sensitivity to specific trace substances.

This must be recognized as a very limited survey which cannot be used for any general conclusions. However, the interviews should provide a basis for follow up action through the health care system to allow the individuals involved to determine if Edmonton water was a source of their difficulties. A leadership role by the Edmonton Board of Health to assist these individuals in resolving their concerns would be appropriate.

8.4 Health Concerns with Microorganisms

A large number of diseases have been documented as waterborne and consequently, the need for reliable disinfection of water is beyond doubt. The need for disinfection is now generally recognized, regardless of the apparent purity of source, because water can become contaminated after it is produced.

The occurrence of waterborne disease outbreaks in Canada and the United States has been rising since the 1950's.[6] This trend is likely due to better investigation and reporting of waterborne disease outbreaks in recent years[7], but the trend indicates that the risks of waterborne disease outbreaks should not be ignored. A multiple barrier concept has evolved within the water industry to provide reasonable assurance that the public will not face unnecessary risks of waterborne disease. That concept should start with the use of a raw water source which experiences minimal microbial contamination. Dr. Smith (report in Volume 2) has documented that the E.L. Smith intake qualifies in that regard, the Rossdale intake does not.

Coliform monitoring is generally used as an indication of fecal contamination of water supplies with routine surveillance. Although positive results for coliforms in treated water provide a reliable basis for followup action, negative results do not assure that the water is free of enteric pathogens.[7] Such assurance must be provided by strict adherence to the multiple barrier concept supported by appropriate monitoring programs.

Dr. Tobin reviewed the bacteriology monitoring data for the Edmonton water system and concluded that it met the Canadian Drinking Water Guidelines[1] for bacterial quality, but there were indications of stressing of the bacterial removal capabilities of the Rossdale plant at certain times of the year. Dr. Tobin also expressed concern over the possibility of under-reporting with the standard plate count monitoring technique employed. More sensitive monitoring of this parameter was recommended because the organisms which should respond to this analysis can include opportunistic pathogens which can infect susceptible individuals.

Dr. Tobin observed that relying strictly on chloramines for disinfection, particularly at the Rossdale plant where adequate contact times could not be provided, was a source of concern. Although a recently completed 50 ML storage reservoir will provide longer contact times, assurance of maximum contact times would require operational practices to be constrained to keeping the reservoir full at all times. Doing so would limit the reservoir to "dead storage" which would reduce operational flexibility in the face of high water demand periods. Reservoir operation, as it relates to assured disinfectant contact time will have to be reconciled with operational needs after site-specific disinfection criteria (section 7.5) have been developed.

Dr. Payment reviewed the circumstances concerning possible viral contamination of Edmonton water and concluded, in the absence of any usable monitoring data on viruses, that the presence of viruses in treated water at Rossdale must be considered possible until proven otherwise. Dr. Payment has monitored treatment plants in Montreal using similar coagulation, filtration and disinfection treatment and found viruses in the treated water. A pessimistic estimate could reach 1 to 10 viruses per 1000L of treated water. Because viruses have a low infective dose (very few viruses necessary to initiate infection) such a level could result in sporadic cases of intestinal disease. These might not be recognized as a waterborne outbreak among all the other causes of gastrointestinal upset. This possibility can only be conjecture in the absence of any monitoring to document the effectiveness of the Edmonton treatment processes for virus removal.

In general, good turbidity removal will provide good virus removal. Dr. Payment has raised concerns with the effectiveness of the winter operating mode which involves flocculation at pH values above 7 (performed only at E.L. Smith but not at Rossdale). Such conditions are not optimum for coagulation and will, consequently, give sub-optimal turbidity removal. Finally, Dr. Payment was also concerned that chloramines alone may not provide adequate assured disinfection with regard to viruses and chloramines would require long, assured, contact times.

Dr. Logsdon reviewed the risks associated with *Giardia* contamination of Edmonton water and, in particular, reviewed the circumstances reported for the *Giardia* outbreak during January to March 1983, when about 550 cases of giardiasis were reported. He noted recent work (discussed in section 6.3) reporting *Giardia* cysts in combined sewer overflows. These findings, together with having 7 combined sewer overflows upstream of Rossdale, place the current

Rossdale intake at risk from *Giardia* challenge. There is also some concern with *Giardia* cysts occurring in dog feces which would contaminate any of the 85 storm sewer outfalls upstream of the Rossdale intake. Thus, although monitoring performed from December 1983 to May 1984 failed to find cysts at Rossdale, the circumstances of the intake being vulnerable to so much potential fecal contamination indicates that the plant must be equipped to withstand a *Giardia* challenge, something it was not judged to be in 1983.[8]

Dr. Logsdon reviewed the epidemiological study[9] performed on the 1983 giardiasis outbreak and disagreed with conclusions that clustering of cases around the Rossdale plant could not be related to increased contact time with chloramines at more remote locations. He notes that chloramines will kill cysts if given enough contact time and the clustering of cases may well be significant.

Dr Logsdon also questions estimates of a requirement for between 1000 and 10,000 infected humans or animals discharging cysts to the river to explain enough cysts passing through the Rossdale plant to cause the outbreak. That estimate of a need for massive river contamination was used to characterize a waterborne outbreak as unlikely. The estimate was based upon providing an infective dose in every liter of treated water delivered for one day. That level of contamination would only be necessary to explain an outbreak affecting most Edmontonians, not an outbreak which affected less than 0.1% of the population.

Finally, and perhaps most important as a lesson for the future, Dr. Logsdon questions the conclusion that there was no difference in treated water quality between the Smith and Rossdale plants. Meeting the current turbidity guideline[1] of 1 NTU at both plants would not insure that the Rossdale plant, which may have been under *Giardia* challenge from an urban sewer source, could not be contaminated with *Giardia* cysts. Waterborne giardiasis outbreaks have been documented elsewhere with water meeting 1NTU for turbidity. The importance of looking beyond guideline values when additional circumstances require explanation is apparent. In summary, Dr. Logsdon notes that because the outbreak occurred in 1983, we can now only offer opinions on its cause. However, we must be careful not to avoid the lessons for the future which that episode may hold for us because the possibility that the 1983 outbreak was waterborne cannot be ruled out.

8.5 Aesthetic Considerations

A brief review of the history of taste and odor problems with Edmonton water is provided in the Hrudey-1 report (in Volume 2). Unfortunately, there was limited documentation available to clearly establish the likely cause of specific taste and odor conditions. Chlorinated phenols had been blamed for many of the problems but analytical data showing specific chlorophenolic compounds in water simultaneously judged to have a medicinal taste were not available. Use of chlorine dioxide since 1982 has been credited with eliminating the chlorophenolic problem, but the lack of analytical data makes this difficult to objectively assess.

Hydrocarbon odors have been a common problem at the Rossdale plant. Recent monitoring data for problem events have identified a wide range of hydrocarbons known to be odourous at very low concentrations. This problem is predominant at Rossdale because of the hydrocarbon inputs it receives from storm sewer drainage and the occasional hydrocarbon leaks at the adjacent power plant (Rossdale uses heated water from the power plant in winter).

Musty odors have been noticeable in raw water from time to time and commonly in the spring. These are associated with microbial byproducts, such as geosmin and methyl isoborneol, which can be odourous at extremely low levels.

Finally, March 1985 and 1986 exhibited odor problems with a different character than had been previously encountered. One unique feature was the occurrence of intense odor from the E.L. Smith plant which had been previously somewhat immune to severe odour problems.

Given this range of odour problems, Dr. Snoeyink and Mr. Trussell both stressed the need to properly characterize the odour causing agent(s) in order to work out effective solutions. For example, Dr. Snoeyink noted that geosmin and methylisoborneol are readily removed by activated carbon adsorption but poorly removed by oxidation. He also noted the need to identify whether the problem arose: directly in the raw water, as a result of reactions at the treatment plant or because of reactions in the distribution system. Again, different approaches would be necessary depending on the answers.

These approaches were presented clearly and concisely by Mr. Trussell. Considering the range of possible contributing factors, he noted the need for thorough investigation in order to be assured of valid solutions. When provided an opportunity to assess the odour of raw and treated water samples from early March, after refrigerated storage for 3 weeks, Mr. Trussell was able to clearly differentiate the raw water which he described as musty/earthy and grassy from the treated water which he found to have a slight sweet and moderately spicy odour. Even in late March, Mr. Trussell was able to detect a musty odour which he characterized as similar to geosmin.

Samples of raw and treated water had been sent to Dr. Brian Brownlee at the National Water Research Institute in Burlington in early March for geosmin and methyl isoborneol analsysis. He found (Brownlee report, Volume 2) traces of geosmin at just above his detection limit of 10 ng/L which is in the detectable odour range. This is about the level estimated by Mr. Trussell based solely on sensory evaluation on March 26. Most Edmontonians would likely find those levels undetectable compared with the peak odours we experienced. As well, these compounds do not explain the type of odour most Edmontonians experienced in early March 1986 and middle to late March 1985.

In reviewing some of the 1985 monitoring data, Mr. Trussell noted the presence of three short chain aldehydes: 2-methyl propanal, 2-methyl butanal and 3-methyl butanal. He suggested that these compounds may be the previously unidentified peaks noted on volatile organics monitoring by the Edmonton water labs. This suggestion was pursued by the Edmonton labs by obtaining the authentic pure compounds. When run on their gas chromatograph, they did appear at about the correct retention times. More important, however, was GC/MS analysis of a March 3, 1986 water sample from the distribution system. This contained substantial quantities of these three aldehydes. Literature evidence indicated that these compounds have very low odour thresholds and measured concentrations in the March 3 sample were 10 to 50 times above reported average threshold odour levels. The World Health Organization[4] suggests that it will normally be necessary to reduce odourous compounds to less than 10% of their threshold odour levels to avoid complaints from sensitive members of the population. These compounds are not likely to be noticed in routine GC/MS monitoring because their predominant fragment ions occur at a lower mass than many laboratories routinely use as a lower cutoff for their mass scanning.

Further corroboration of these compounds being involved in the 1985 and 1986 events came from preparing a cocktail of the compounds and soliciting opinions on the similarity to what inidviduals recalled for the March water. Many people recognized it immediately. Some were not sure or could not recall the March odour. These compounds are known to have very complex odours which strike different people in different ways. In the case of 2-methyl propanal, a large percentage of odour panelists were found to be very insensitive to the odor.[10]

In summary, these aldehydes seem to qualify as likely candidates for causing at least part of the 1985/86 problem. However, the original advice from Dr. Snoeyink and Mr.Trussell to properly characterize the problem must be recalled. Although the identification of these compounds provides a useful lead there are many unanswered questions about how they arise and, consequently, how they can be avoided in the future.

8.6 Overall Assessment

The performance of the Edmonton water treatment plants in producing adequate quantities of treated water which consistently meets the Canadian Drinking Water Guidelines[1] is excellent. Edmonton water contains very low levels of those substances which can be reliably assessed for health effects .

Concerns for potential microbial contamination of treated water have been expressed primarily based upon expert opinion, because confirmatory evidence of specific problems in this regard is not available. However, a water utility must act on reasonable suspicions concerning health-related problems, particularly when offered by an experienced group of water experts. Recommendations for better turbidity removal and development of site specific disinfection criteria will address these concerns

The occurrence of taste and odour problems in Edmonton does not require expert evidence for confirmation of occurrence. However, the expert interpretations offered do point to probable causes. With a commitment to pursue the leads identified, the overall nature of taste and odour problems with Edmonton water can be understood and appropriate, cost effective solutions should be possible.

8.7 Specific Recommendations

1. The Edmonton Board of Health should prepare a concise review of the demonstrated health benefits arising from fluoridation of our water supply which can be made available to Edmontonians concerned about the value of this practice.

2. The Edmonton Board of Health should communicate with the physicians of the eight individuals interviewed by Dr. Rogers to offer assistance in determining whether Edmonton drinking water might have played a role in their health problems, or to arrange further evaluation if appropriate. Sampling programs directed at the indviduals' home water supply should only be developed in response to identified problems.

3. In keeping with the concerns raised by Dr.Bull and Dr. Rogers, the bulk chemical and materials testing program should be used as a starting point to review the distribution system and common plumbing system characteristics to develop a distribution system monitoring program for targeted substances. A general survey scanning all available routine parameters will consume massive analytical resources for each sample when most parameters are likely to be irrelevant. Specialized parameters like targeted GC analyses (using MS only for verification), purgeable organic carbon and specific metals are more likely to be useful for this type of survey.

4. If experience over the next year indicates that available treatment processes cannot be expected to control organic loading from rural runoff, a progressive series of toxicological evaluations, as recommended by Dr. Bull will become necessary. If substantial success is achieved in eliminating the problems, the need for such testing will become academic for Edmonton. This view is predicated on the premise that solutions can be found for non-odourous organics as well as odourous ones. Simply eliminating the latter, may not eliminate the basis for concern.

5. If monitoring for *Giardia* , or some other explicit justification, leads to a long term commitment to chlorine dioxide use, then the epidemiological studies suggested by Dr. Bull should become a high priority.

9. SIGNIFICANCE OF DRINKING WATER TO THE HEALTH OF EDMONTONIANS

This assessment will draw extensively from the report (Volume 2) by Dr. Richard J. Bull, Associate Professor of Pharmacology / Toxicology at Washington State University. Dr. Bull was given the task of considering the health-related information and relevant reports by other experts to formulate an overall opinion on the matters of health. Dr. Bull was assigned this role because of his extensive background in water-related toxicology matters (see Appendix B). That background involves 22 years of toxicology experience including 14 years with the U.S. Environmental Protection Agency, Health Effects Research Laboratory in Cincinatti.

Dr. Bull outlined the basis for his assessment which was necessarily limited by time, resources, the scope of information which had been gathered on Edmonton water and also by the general state of our knowledge on some issues.

> *"It should be understood that there are a great number of unknowns involved in judging the extent to which drinking waters might present subtle toxicological hazards to the consuming population. Most of these problems are by no means unique to Edmonton. First, we are limited by our analytical capabilities; only a small portion of the organic material derived from surface sources can be chemically identified. Much of this material is simply natural organic matter with varying and complex structure. This natural organic matter forms a precursor pool that reacts with added disinfectant to form by-products that are of some toxicological concern. A second problem is that an appropriate toxicological data base does not exist for many of the chemicals that have been identified in drinking water. This is particularly true of reaction products of disinfectants because some of these compounds are not produced commercially. These are very difficult and important problems that require further research. However, this lack of information should not be viewed with undue alarm. The information which is available (e.g. epidemiological data) would suggest that any additional hazard that might be associated with these variables is small, perhaps non-existent."*

Dr. Bull organized his assessment into six categories: I.mutagenicity studies, II.epidemiology information, III.review of toxicology of specific compounds, IV.examination of treatment processes for health issues, V.examination of proposed research studies, VI.summary and conclusions.

I. Mutagenicity.

Dr. Bull reviewed a series of mutagenicity studies on Alberta waters. Mutagenicity is a characteristic indicating the ability of a substance to cause genetic mutations. Because cancer causing agents (carcinogens) involve genetic alteration in their mode of action, mutagenicity is an important characteristic. Dr. Bull cautions:

> *"There are severe limits on how results from mutagenicity testing done in in vitro systems can be applied to estimating human health hazard."*

This is a concern because a critical component of carcinogen assessment is the potency of the chemical. In other words, how little of the chemical is required to cause an effect. A highly potent carcinogen cannot be tolerated at even very low levels. A weak carcinogen may not pose any significant risk at realistic levels of exposure. The critical point is that you cannot infer potency of a chemical strictly from mutagenicity tests done with bacteria. Potency data must be developed from animal studies wherein a range of doses is used to determine corresponding response.

Dr. Bull also notes that mutagenicity tests are developed to produce a minimum number of false negative responses (i.e., no mutagenic response observed for a true carcinogen), because they are intended for a screening role. In this function, we do not wish to have the screening test reject chemicals which really warrant further testing. Accordingly, these tests also have a high false positive rate (i.e., measured mutagenic response for a non-carcinogen), working with pure chemicals. The likelihood of false positives with mixtures of chemicals is even greater.

In summary, Dr. Bull found that:

> *"Edmonton residents can take some comfort in the fact that there is little evidence that mutagenic chemicals are present in their finished drinking water. This is primarily attributable to the use of chlorine dioxide as a disinfectant rather than chlorine."*

II. Epidemiological Studies.

Dr. Bull explains that, ideally, the best data for health assessment would be based on human exposure. Such data could be obtained by human experiments or studies of existing exposure circumstances, occupational or otherwise (epidemiology). Obviously, human experiments are rarely possible for substances with potentially severe health effects. Consequently, for data directly relevant to man we are usually limited to epidemiological data. In those cases where such information is available, it is extremely important to informed decision-making. However, there are also definite limits to the application of epidemiology for risk assessment.

The human exposure is not controlled and often difficult to even estimate. With infectious disease, finding of an agent which can be shown to initiate the infection provides strong evidence for causality. With chemical agents, their effects are usually not highly specific and it is generally much more difficult to assign causality. Negative epidemiological results, those showing no evidence of an effect, are often encountered. These are difficult to interpret without knowledge of the limits of sensitivity (what is the smallest effect which could be detected by the sample size and population variablity encountered?). After noting that :

> *"There appears to very little epidemiological data that is available and relevant to the quality of Edmonton's drinking water."* ,

Dr. Bull states:

> *"some comfort can be taken from the fact that there are no unusual patterns of chronic disease mortality that could reasonably be associated with drinking water quality."*
>
> *"In the final analysis, however, one must take the conservative view that there is no epidemiological evidence that Edmonton's water supply has contributed adversely to the public health."*

III. Review of Individual Chemical Substances.

Dr.Bull first considered inorganic substances as indicated by the City of Edmonton 1984 Annual Report and the Alberta Treated Water Surveys from 1983 to 1986. In general, there was not too much noteworthy with the inorganics. A mercury result for 1984 which marginally exceeded the maximum acceptable concentration was noted in the City's Annual Report, however, the average value was well below the standard. This was interpreted as being primarily a case for followup to identify the cause rather than an issue of significant health concern. The only other comments on inorganics were reserved for aluminum. The case for controlling aluminum which was also made by Dr. Logsdon and Dr. Snoeyink was based principally on process control factors rather than documented health effects.

Review of the organic substances was prefaced by a general observation.

> *"organic chemicals that were identified in routine analysis of Edmonton's drinking water were present at such low levels that would be of little concern on an individual basis. In a sense, total reliance on a chemical-by-chemical approach to define a toxicological hazards in drinking water avoids the basic question of whether the water is safe or not because such a small fraction of the organic material present in drinking water sources can be identified and measured (estimated to be less than 1%). Again this is* *not* *a problem that is peculiar to Edmonton and is one that cannot be simply resolved. However, there are some patterns to the contamination that should be taken into consideration. It is not necessary to have all this information to come to some remedies that avoid potential problems by the exercise of common sense."*

Dr. Bull suggested there was evidence for three patterns of organic contamination:

a.chemicals arising from urban runoff,

b. chemicals which are associated with the spring rural runoff,

c. chemicals which are routinely found.

With regard to the urban runoff, Dr. Bull discussed xylenes as an example of chemicals which may reasonably be assumed to arise from the storm sewer outfalls upstream of the Rossdale intake. Although the toxicology for these specific chemicals does not provide a cause for concern , Dr. Bull asks:

> *"what other chemicals might come from this source that would be undesirable contaminants of drinking water....The choice appears to further document and study the nature of that contamination and its health effects or to eliminate the source....Common sense would suggest that removal of factors which make the system vulnerable might be the most cost-effective approach in the long term."*

For the chemicals which appear routinely, Dr. Bull concludes:

> *"there is again little to fear form a health effects standpoint. The trihalomethane levels are lower than would be expected in water supplies that use chlorine as the sole disinfectant."*

Even by considering the chemicals which appear rarely, Dr. Bull finds limited direct cause for concern.

> *"Non-carcinogenic chemicals identified are present at such low concentrations that no adverse health effects could reasonably be anticipated. While there were trace quantities of chemicals that are putative carcinogens, the risk levels that can be projected using a worst case analysis are vanishingly small. To put this in context, the estimate of risk that would be derived by adding up all the risks indicated above would be approximately an order of magnitude less than the risk permitted by the WHO guideline for chloroform in drinking water. In general, one would have to conclude that the levels of risk that would be anticipated from the organic chemicals identified in Edmonton 's water is considerably less than would be calculated for most cities that use chlorine for the purposes of disinfecting drinking water."*

Finally, Dr Bull deals with the chemicals associated with the spring runoff which others have attributed to rural sources. These organics can also interact with the oxidant , chlorine dioxide to generate new chemicals or increase concentrations. This group is the hardest to deal with because they are so poorly documented. Dr. Bull noted the results of the non-routine analysis by Dr. Coutts (Hrudey-1 report, Volume 2) which identified a wide range of organic products which might not appear in more routine analyses. Given our inability to assess these chemicals specifically there is a fundamental case for seeking to either prevent their formation or to eliminate those that inevitably form.

IV. Potential Contributions from Drinking Water Treatment Processes to Adverse Health Effects.

Dr. Bull discussed some problems which may arise from disinfection practices. He notes the difficulty experienced in trying to maintain a residual of chlorine dioxide which would be adequate for *Giardia* inactivation during the spring runoff demand and the resulting recourse to higher dosages. The experience at Newport News, Virginia, previously noted by Dr. Snoeyink was cited as a case of consumer complaints of eye and nose irritation at chlorine dioxide doses above 1 mg/L. The difficulty faced in trading off these problems with those likely to be caused by using chlorine and the concern about the adequacy of chloramines for *Giardia* were all reiterated.

Because the problems associated with excess chlorine use are well known and the practice in Edmonton involved use of the less well known chlorine dioxide, Dr.Bull devoted attention to the toxicology of chlorine dioxide itself and that of its principal inorganic byproduct, chlorite. Using common approaches to the treatment of experimental data he arrives at objective maximum levels of 0.13 mg/L for chlorine dioxide and 0.10 mg/L for chlorite. These values represent maximum levels for residual concentrations after contact time between the chlorine dioxide and the water being treated.

By comparison, the only published policy by the U.S. Environmental Protection Agency[11] was provided in 1978 and it appears to limit the dose of chlorine dioxide to 1 mg/L, which would normally limit the residual chlorite to about 0.5 mg/L. Depending upon demand exerted by substances in the water, this maximum dosage level might leave no residual chlorine dioxide in the water.

Dr. Bull explains his calculations as follows:

> *" The above exercise is consistent with USEPA policy, but does not necessarily mean that these are the levels that will be chosen as standards for chlorine dioxide or chlorite. Although some rationale is articulated for the safety factors of 10 and 100 used above, the selection of these values is somewhat arbitrary. Consequently, the Agency could depart from these levels if they feel it can be justified.*
>
> *I ran through this exercise for two reasons. The first is to document that there is an independent component to the toxicities of chlorine dioxide and chlorite suggesting that independent standards should be established (i.e. the thyroid effect appears independent of the hemolytic effect). Of course the hemolytic effects of the two agents are probably not independent, but by the time one calculates the conversion of chlorine dioxide to chlorite vs. other species, the incremental risk would be quite small. The second reason is to impress upon the people in Edmonton that there need to be genuine limits on the amount of chlorine dioxide that should be added to drinking water. The actual number*

established as the limit depends on the chance one is willing to take that an ***acute*** *illness will result. I emphasize the word acute because the effects we are worried about with chlorite are acute hazards that will be induced by short term excursions over a critical dose. It is not a case where average doses over a lifetime is important as is presumed the case for chemical carcinogens. I will also point out that there is no question that humans are susceptible to agents that produce hemolytic anemia so the question of whether the effects extrapolate across species or not is not an issue. To put this in perspective, the same cannot be said for trihalomethane-induced cancer. In the case of the anti-thyroid effects of chlorine dioxide, the effects of concern would have to be exerted over some time period to result in irreversible effects. In the developing human, the critical period of exposure (during pregnancy or in the postnatal period) could be relatively short (weeks or months) whereas in the adult thyroid function would have to be depressed for some time (months to years) before an irreversible effect would become manifest (e.g. thyroid cancer would be a possibility).*

For the above reasons, I was very happy to see that Edmonton waterworks is now placing considerable emphasis on the measurement of chlorine dioxide and chlorite residuals in the water provided to the consumer. The movement of the point of chlorine dioxide application downstream would also appear likely to decrease the dose of disinfectant required. However, there appears to be some conflict between the amounts of disinfectant that are needed to protect the system against contamination by Giardia and the need to minimize the dose of disinfectant from the standpoint of producing toxicological problems."

Finally, Dr. Bull stresses the need to focus attention on the quality of bulk chemicals and construction materials used in the systems.

V. Recommendations for Research in the Health Effects Area.

Dr. Bull reviewed the circumstances as he could determine them and made specific recommendations concerning aspects which require further research. He subdivided these into those questions of peculiar concern to Edmonton and those of broader national or international concern. These will be dealt with in the recommendations section of this report.

VI. Summary and Conclusions.

Dr. Bull provided the following summation:

> *"It is obvious that Edmonton has periodic problems with the quality of its drinking water. As unpleasant as that problem is to the consumer, there is little objective reason to suspect that the chemical contamination associated with these episodes has produced any adverse effects on health. This conclusion is, of course, qualified by the limited amount of information that is available that bears directly on the issue. On the other hand, is should be pointed out that the information that has been assembled for evaluation of Edmonton's water approaches that which is possible within the limited period of time available. It is important that work begin to address some of the obvious ills in the system based upon the information that is available. At the same time, I hope that Edmonton accepts the responsibility for better characterizing their source water and their treatment of it so that as problems arise in the future there is an objective data base that can be used to solve these new problems.*
>
> *Although it is not possible to point to individual chemicals at the concentrations encountered in Edmonton drinking water as being responsible for adverse health effects, there are several situations which exist in the Edmonton system that could easily allow chemicals and/or microbial agents into the system at concentrations that are sufficiently high to produce adverse health effects. Of these, inadequate control of the disinfectant application is undoubtedly the most critical and in my view the most likely to produce adverse health effects if not strictly controlled. The remaining areas also carry some risks, but they are more reflective of good water treatment practices that protect the water from unanticipated problems rather than being addressed as a particular health effects issue."*

10. Options for Improvements

10.1 Upstream Pollution Control

A review of water quality monitoring data going back to 1963 and consideration of the extent of municpal, agricultural and industrial activity upstream of Edmonton leads to a conclusion that there is minimal scope for any water quality enhancement by means of stricter upstream pollution control. The general water quality of the North Saskatchewan River as it arrives at Edmonton is good. The exceptions to this generalization, periods of high turbidity and dissolved organic carbon cannot be blamed upon upstream activities, as far as we can determine from our current understanding.

The current practice of requiring municipal effluent disinfection at Devon and Drayton Valley is prudent and should be maintained. Steps to reduce inputs from indirect seasonal discharge municipal lagoons which can be connected to regional sewerage systems are worth continuing.

A regional inventory of bulk chemicals handled in the upstream watershed would be useful for spill contingency planning. Cooperative association with upstream users of North Saskatchewan River water would also be beneficial in responding effectively to unusual river conditions which may be noticed. Experience to date indicates that the City of Edmonton has been notified by Alberta Environment in cases of reported spills or unusual water conditions on the river.

A policy of strict control of any proposed new upstream discharges to the North Saskatchewan River must be developed. Given all the limitations which are inherent in our monitoring and treatment systems, a decision to allow any significant waste discharges upstream of Edmonton would be unwise. The best protection available for raw water quality is still to limit the inputs which may adversely affect that quality . With 30% of the population of Alberta dependent upon the North Saskatchewan River for its raw water source, it is difficult to imagine an industrial project having sufficient net economic benefit to Albertans to warrant compromising the safety currently provided by upstream water quality.

Within the City of Edmonton, water quality for drinking water abstraction is degraded by the input of 85 storm outfalls upstream of the Rossdale intake. The option exists to collect the discharge of all outfalls and pipe them downstream of the Rossdale intake. Alternately they could be collected to common points and subjected to treatment. Those options are not cost-effective compared with the option (section 10.2) of moving the raw water intake upstream of the influence of the storm outfalls.

Reasons for this judgement may be summarized as follows.

1. Moving the discharges of all 85 storm outfalls downstream of the Rossdale intake will necessarily be more expensive than moving the one intake upstream. No improvement in river water quality downstream of Rossdale would be achieved for the extra expense involved.

2. Providing treatment of all storm discharges to the degree necessary to deal with the expressed concerns about raw water supply would require Edmonton to undertake an exercise of a scale and scope well beyond that performed by any other large municipality in North America for storm water control. Reliable cost estimation would be difficult given the lack of prior experience with such an undertaking. The overall effects of storm discharges on general river water quality have not yet been shown to warrant that level of undertaking, although the poor quality of the North Saskatchewan River for water-contact recreation has been clearly documented. That problem should be improved by the current efforts to eliminate combined sewer overflows upstream of Rossdale.

3. The overall question of what level of water quality Edmontonians wish to have, and are willing to pay for, in the North Saskatchewan River is an important decision which should be addressed. However, the basis for sound decisions remains to be developed and the issues are as much social and political as they are scientific. The resolution of the raw water supply at Rossdale is too important to delay action while the broader questions of river management are being resolved.

4. Leaving the Rossdale intake in the center of Edmonton will still leave it more vulnerable to spills arising from urban actitivities. Human activities upstream of Edmonton are very limited by comparison.

Considering all these factors, and the direction of this study to focus on the requirements for a safe water supply, moving the Rossdale intake upstream is clearly preferable.

10.2 Water Intakes and Storage

The location of the Rossdale water intake has been unanimously criticized by all the experts. The water quality monitoring program performed by the City of Edmonton during the spring of 1986 has verified the fluctuations in water quality imposed upon the Rossdale plant by urban runoff. Previously documented circumstances of storm runoff impact, spills and microbiological contamination impacting the Rossdale intake have been highlighted throughout the expert reports. The spring 1986 monitoring program also showed that other intake locations further across the river, at the Rossdale cross-section, would not provide adequate improvement over the existing north bank intake.

These factors were subjected to modelling by Dr. Smith and associates (Smith report, Volume 2). That exercise clearly established the vulnerability of the Rossdale intake to influence from upstream storm sewer inputs. Modelling was done using bacterial indicators but the results could be applied to chemical contaminants as well. There appear to be locations upstream of Groat Bridge which, for an intake in the center of the river, water quality would not be influenced by storm sewer discharges. The critical discharge for siting a new intake would be the Mill Woods outfall which is located in Terwillegar Park just downstream of the E.L. Smith plant.

The objective for relocation of the Rossdale intake should be to reliably obtain water which is equivalent in quality to that available at E.L. Smith. Moving the intake all the way up-river to the E.L. Smith intake would guarantee achieving that goal. The modelling studies by Dr. Smith suggest that this may be achievable at a location between Groat Bridge and the E.L. Smith plant. The exact location for such an intermediate solution can only determined by detailed engineering studies with appropriate field monitoring to verify the model predictions. Because the large diameter pipes required for the intake pipeline are very expensive, siting studies are likely to be cost-effective. An intermediate location must only be accepted with substantial assurance that the new intake will be unaffected by upstream storm sewer inputs for all reasonable flow scenarios, both present and future.

The possibility of offstream storage as an expedient for protection against spills and poor water conditions in the river was explored in a 1983 study.[12] This determined that provision for 11 days storage would be adequate to deal with a hypothetical spill scenario arising at Rocky Mountain House. The 11 days would also appear to be adequate to deal with the peak of dissolved organic carbon which was experienced this year. There is inadequate detail in the records for organic carbon from previous years, but peak values can be inferred from color records. These suggest that 10 to 15 days would cover most peak episodes based upon 1981 and 1982 data.[12] The concept of offstream storage is very attractive because winter water quality is excellent and spring and summer quality is very manageable given some lead time to adapt the process. The major difficulty with this option is finding a suitable location for a reservoir of the necessary size. A total of 7 alternate sites were evaluated with cost estimates. The most cost effective of these was included in our cost evaluations in section 10.5.

Although recommendations to pursue the storage option will be deferred until the impact of Rossdale intake relocation planning and process upgrading can be assessed, the possibility of pursuing this option in the future should not be ruled out. Planning for other actions should, therefore, not preclude a future decision to proceed with raw water storage. One attractive intermediate option, discussed by Prof. Bernhardt, is the possibility of short term flow-through storage which could be used as a pretreatment basin at times of poor raw water quality.

In summary, relocation of the Rossdale intake to achieve better raw water quality is necessary. Providing a raw water supply which will insure the best protection of public health reasonably possible is a fundamental principle. Relocation of the intake will provide several tangible benefits including:

1. a guaranteed, dramatic reduction in the risk of treated water contamination by microorganisms and chemical contaminants

2. reduction in short term fluctuations in raw water quality will allow better prospects for process optimization which may save money on chemical use and will insure better treatment performance for the removal of chemical and microbial contaminants.

3. a substantial reduction of the existing vulnerability of the Rossdale intake to a toxic spill event within the city.

The arguments in favor of this action are widely recognized, as indicated by this quote from the U.S. National Academy of Sciences landmark publication, Drinking Water and Health.[13]

> *"Good engineering and public health practices emphasize the need for using raw water of the highest possible quality. Bacteriological testing, or the imposition of bacteriological standards, are adjuncts to, not substitutes for, good-quality raw water, proper water treatment, and integrity of the distribution system."*

The validity and recognition of these principles is not new as excerpts from a 1950 consulting engineering report[14] to the City of Edmonton concerning its water system illustrate:

> *"The location of the intake on approximately the north and south centre line of the city is economical from the standpoint of water distribution, but not so favourable for water quality. In general water works intakes should be located upstream from the community in order that the water will not be polluted by the drainage from adjoining lands.*
>
> *The maintenance of the water works intake in its present location means that all sewage must be delivered downstream. With the growth of the city in the westerly direction this involves a large additional investment in the sewerage system, and continuous vigilance on the part of the city to prevent pollution getting into the river upstream from the intake.....The greatest care should be exercised to insure that no septic tank effluents, sanitary sewage or other pollution reaches the river upstream from the intake."*

This report was written when Edmonton still employed only a combined sewer system. The need for separating the sewer system upstream of the Rossdale intake was clear. The report recommended, assuming that sanitary conditions would be maintained to keep storm drainage from conveying pollution:

> *"...it is definitely recommended that the existing location be maintained and that all precautions be taken to eliminate upstream pollution."*

The caveats placed on this 1950 recommendation have not been met because of the combined sewer overflows and cross-connections which occur upstream of Rossdale. Furthermore, we now have a better understanding of the pollution loading which arises from separated storm sewers.

10.3 Process Evaluation and Modified Treatment Technology

A wide variety of process options were proposed by various experts in varying levels of detail. None of these proposals would be recommended for implementation without considerable bench scale evaluation. For the more radical alterations, pilot scale testing will also be necessary.

Without having had the opportunity to compare the various proposals with some bench scale testing, it is not possible to make choices of which will be most cost effective for implementation. Furthermore, the comparative need for some options is contingent upon the speed with which the Rossdale raw water quality can be improved by intake relocation.

However, some generalizations are possible. Firstly, all experts agree on the need for thorough process evaluation by a specialist "process improvement team" before implementing any significant change to treatment. With a competent process improvement team in place, the existing processes can be adjusted to variations in raw water quality on a continuing basis to fine tune treatment for best performance. The ability to evaluate and adjust treatment conditions to those best-suited for given raw water conditions can result in savings in treatment chemical costs and should provide more consistent and better overall treatment performance using existing processes.

There is general agreement on the need for improved turbidity removal with a goal of consistently meeting 0.1 NTU as the objective for process improvements. Substantial improvements should be possible with existing facilities operated under optimum chemical dosage conditions. Consistent achievement of this goal may require facility expansion to reduce process loadings and/or provide new process steps.

There is general agreement on the need to optimize organics removal at times of high dissolved organic carbon to minimize the dosage of oxidant required to achieve a disinfectant residual. Failure to do this will result in unnecessary formation of oxidation byproducts both organic and inorganic. Optimized organics removal will require much better characterization of the raw water organic carbon. A demonstration of potential in that regard is a preliminary evaluation by the Alberta Research Council of the humic materials in the March raw water by a variety of state-of-the-art analytical procedures (Hrudey-1 report, Volume 2).

There is general agreement that disinfection practice needs to be carefully assessed. No clear solution to the disinfection dilemma has emerged from the various evaluations. That arises, in part, because the extent of risk at E.L. Smith is not clear, whereas, expedience allows assumption of risk for Rossdale. Without a clear understanding of the degree of likely pathogen challenge at E.L.Smith , it is difficult to be certain about the critical disinfection criterion. That does not pose a problem for the turbidity removal goal because there is no downside risk involved other than the challenge posed. The same is not true for disinfection. In this case, more is not necessarily better, given the downside risks associated with disinfection byproducts. Notwithstanding this dilemma, some rationale is put forward in the recommendations to deal with disinfection. The key feature is the need to develop relevant disinfection criteria for Edmonton's specific situation.

There is some agreement on the option of applying enhanced activated carbon usage. This may be through process changes to achieve better use of the powdered activated carbon, replacement of filter media with granular activated carbon or granular activated carbon contactors following existing filters. These are listed in increasing order of difficulty and need for justification prior to implementation. There seems little doubt that activated carbon in some form will remain an essential part of the available process options in the future.

There is some agreement that ozone should be investigated as a future process option when better understanding of the organics involved in treatment has been acquired. Implementation of ozone must include consideration for incorporation of downstream activated carbon to deal with ozone byproducts.

10.4 Point-of-Use Options

The performance and viability of point-of-use water treatment devices was discussed by Dr. Logsdon and Dr. Tobin. Available types were generally categorized into activated carbon, reverse osmosis and distillation devices. Activated carbon is effective at organics and chlorine removal. However, research has shown that the buildup of nutrients on the carbon can promote colonization by heterotrophic bacteria (those which should be characterized by the standard plate count test described in section 6.2). Dr. Tobin quoted values of 10^9 bacteria per liter which have occurred after overnight standing of water in the filter. Thus, carbon filters must be flushed thoroughly after long standing periods and they should never be used to treat a water which is not already microbiologically safe.

Dr. Tobin noted that major advances in reverse osmosis have been made in recent years. This process can be used to remove bacteria, most positively charges ions in water, but removal of negatively charged ions (like nitrate) is poor. Another disadvantage is that they only produce 10% usable water and waste 90% of the water fed.

Dr. Tobin described home water distillers as effective at removing pathogenic microorganisms from water, but the vessel which receives the condensed steam can become contaminated with *Pseudomonas* after long term use. He also noted that many organics of concern in drinking water are volatile and these can be carried over with the steam and be condensed in the distilled water. This to happened with our laboratory distilled water this spring, because the aldehydes discussed in section 8.5 are volatile. Thus, distillers are known to be effective at removing inorganic substances but their performance on volatile organics may be poor.

With regard to evaluation, Dr. Tobin noted that there are several agencies in Canada and the United States who have evaluated various home water treatment devices for their performance. He singles out the National Sanitation Foundation (Ann Arbor, Michigan) as an agency with a major evaluation program.

Overall, Dr. Tobin concludes that activated carbon alone or in combination with reverse osmosis or distillation is most likely to cause improvements in taste and odour. Some devices on the market have attempted to combine processes to overcome known deficiencies in any one. Verifying commercial claims with the National Sanitation Foundation testing program would be prudent.

With regard to the applicability of point-of-use treatment devices to Edmonton's situation, Dr. Logsdon notes that they are best suited to small communites or individual systems. These devices do require maintenance which might be feasible for a utility to provide in a small community. Such would not be appropriate for Edmonton. Dr. Logsdon notes that a failure to deal with aesthetic problems existing with the municipal water supply will encourage increasing numbers of citizens to invest in their own devices to deal with the problem. Substantial investment by individual taxpayers could eventually erode general citizen support for needed improvements to the municipal system.

10.5 Costs of Viable Options

Although it was difficult during the course of deliberations by the various treatment experts, to predict the actions which they would eventually recommend, a list of possible options was provided to the Water Plants Engineering Group of Edmonton Water and Sanitation for the preparation of preliminary cost estimates.

As it has turned out, the only specific action which is clearly recommended for immediate action is the relocation of the Rossdale intake. Other treatment alternatives are contingent upon the results of specific problem characterization and the results of proposed process optimization studies. The relocation of powdered activated carbon (PAC) application seems likely to arise out of these studies. All improvement options still under consideration were reviewed, in any case, to provide an indication of the size of investments which may ultimately be required.

These costs are summarized in Table 1 for those actions which are recommended or are still under consideration. These options do not precisely fit the recommendations. Without the necessary engineering studies, correct cost figures cannot be determined for the cost of intake relocation because the exact location remains to be decided. However, we can determine from Dr. Smith's report that the very minimum acceptable Rossdale intake relocation would be upstream of Groat Bridge at a cost of $11,000,000. The maximum would run all the way to E.L. Smith at a total cost of $46,000,000. Thorough modelling and field verification should be able to reduce costs substantially below the latter figure, unless there are other advantages to Edmonton Water and Sanitation in having the intakes consolidated at the E.L.Smith plant. The influence of intake location on future storm sewer outfall locations will also need to be considered.

Because capital costs in millions of dollars will have little meaning to the average citizen, Edmonton Water and Sanitation developed water utility rate increase scenarios for the average household, given various cost options. These are plotted in Figures 9 and 10. Clearly the impact

Table 1. Summary of Estimated Costs for Specified Improvements

Option	Capital Cost ($1,000)	Annual Operating and Maintenance ($1,000)	Remarks
1.Extend Rossdale intake line upstream of Groat Bridge	11,000	60	includes new intake, piping and pumphouse
2.Extend Rosssdale intake line upstream to E.L. Smith	46,000	80	includes new intake, piping and pumphouse
3.Isolated storage reservoir	122,000	224	includes two pumphouse and piping
4.Post-filtration Granular Activated Carbon (all plants)	112,000	1800	includes new filter building and regeneration facilities
5.Granular Activated Carbon replacement of existing filter media (all plants)	8,300	460	includes regeneration facility
6.Ozonation before filtration (all plants)	24,000	1000	includes ozone generation facility and new building
7. Powdered Activated Carbon relocation (all plants)	6,100	54	includes new contact basin for powdered activated carbon addition
8. Expand E.L. Smith plant	32,000	1,600	addition of 2 clarifiers

Factors used: 1986$; interest during construction of 11.63%; overhead of 4%; engineering fee of 7%; design period of 1986 to 2001 and design flows for both plants of 400MLD

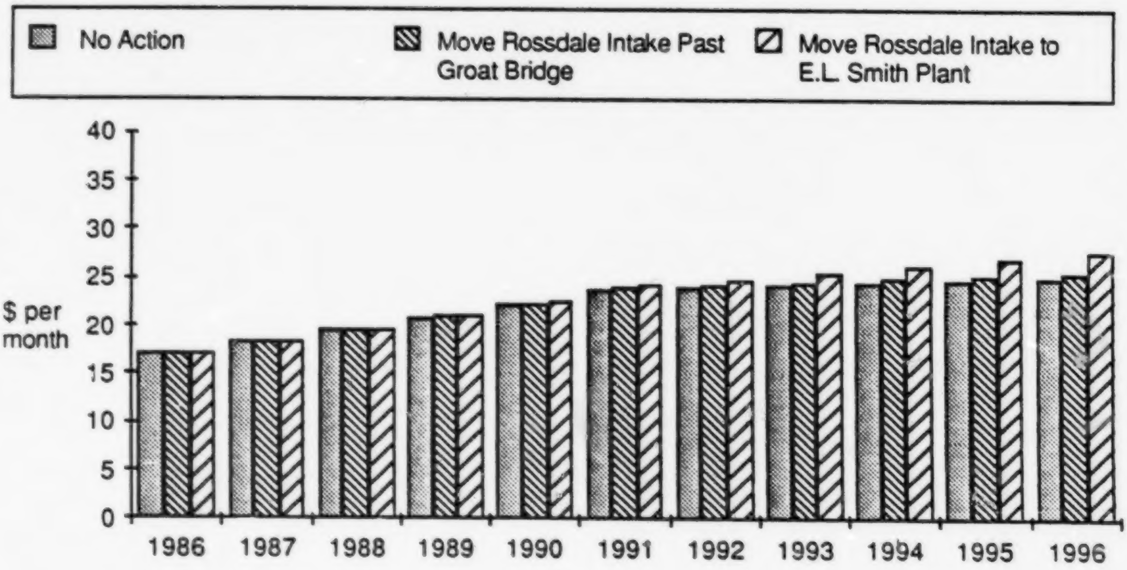

Figure 9. Impact on Average Monthly Residential Water Bill for Intake Relocation.

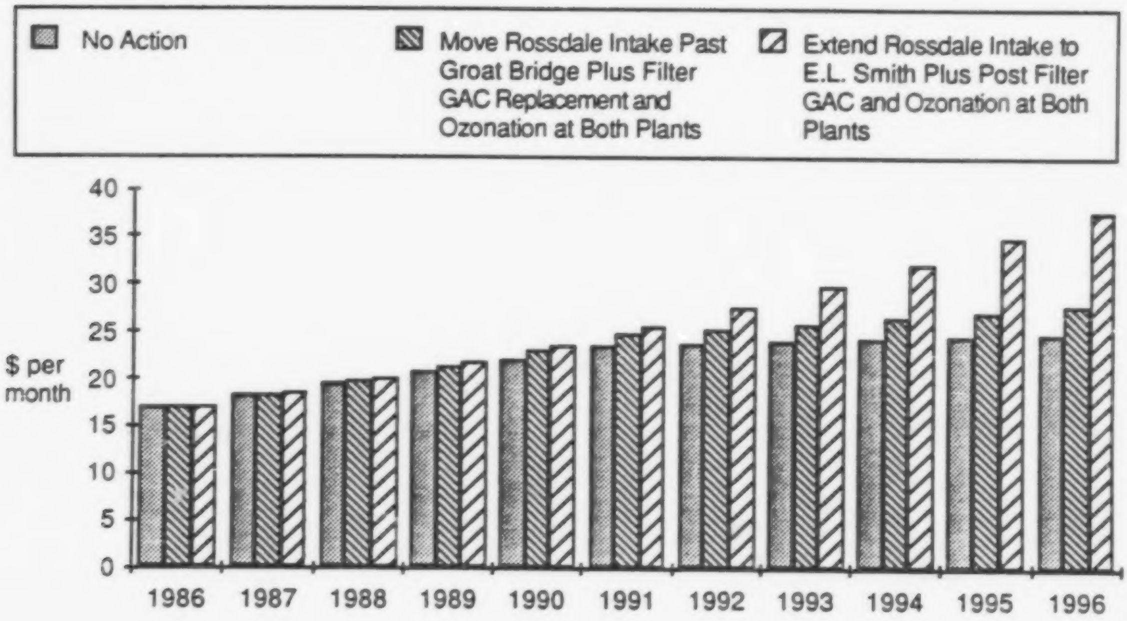

Figure 10. Impact on Average Monthly Residential Water Bill for Intake Relocation Plus Specified Additional Treatment Processes.

of intake relocation (Figure 9) will represent a small portion of the average monthly water bill. These projections include rate increases which will occur without any system improvements. For the combination of all conceivable advanced treatment options (Figure 10) the rate increases are substantial but not astronomical. A more economical advanced treatment scenario involving filter media replacement by granular activated carbon is shown in Figure 10.

11. INTERPRETATION

Section 1 outlined a statement of need which gave rise to this study. Following is an assessment of the study findings as they pertain to the stated need. Three basic questions were posed:

1. How does Edmonton drinking water compare with other jurisdictions?
2. Are there valid health concerns which must be addressed?
3. Are there feasible solutions to the intermittent aesthetic problems which have arisen?

Dealing first with Question 1, evaluation of both raw water sources and treated water quality indicates that, on a normal basis, Edmonton's water compares favorably with that available elsewhere in North America. In general, the treatment processes employed by Edmonton are comparable to other North American cities. There are specific cases of advanced treatment practice in Europe which routinely produce better quality water than normally found in North America. The technologies involved are receiving increasing interest in North America and will find greater application in the future.

A notable exception with Edmonton regarding comparable practice is the location of the Rossdale raw water intake. Although there are many cases where large cities will have to accept poorer quality raw water than provided by the Rossdale intake, this is accepted because of a lack of alternatives, not by choice. Consequently, the experts consulted for this study have unanimously criticized the Rossdale raw water intake location as representing an unnecessary risk and a poor choice for insuring adequate public health protection.

The Rossdale intake situation is a case where strict reliance on meeting the guidelines[1] is not adequate. By analogy, a person may always observe the speed limit and may be certain that his or her blood alcohol levels are always well below the legal limit, but that person should not ignore maintenance advice from a knowledgeable mechanic if his or her vehicle's brake fluid level is too low or its tires are worn. The guidelines are intended to provide assurance of drinking water quality when appropriate source selection and treatment procedures have been used. They should not be used to try and justify continued use of a problem raw water source.

The 52 parameters covered by the guidelines cannot completely represent the more than 7 million chemical substances currently known to man. Furthermore, routine compliance with guideline values provides no reassurance with regard to the vulnerability of the existing Rossdale intake to adverse impact from toxic chemical spills to the upstream storm sewer system.

There have also been concerns expressed by various experts about the capability of the Edmonton water labs to efficiently and reliably monitor the chemical and microbiological quality of Edmonton water. These concerns must be redressed to provide assurance that the conclusions about good treatment performance can be sustained.

Regarding the second question concerning health issues, the public would like a yes or no answer to the question: "Is it safe?" Naturally, they are frustrated when they hear an answer of: "Yes, but......" Because of the nature, complexity and uncertainty of the issues, that answer is probably the best which can ever be given. However, perhaps a more relevant question needs to be asked, that being: "Should I be concerned?"

Dealing with Edmonton water, this study has found that for all the substances for which there are health-related values specified in the Canadian Drinking Water Guidelines[1], Edmonton water is safe and there is no cause for concern. In addition, for those trace organic substances, including carcinogens, which have been adequately studied for their toxicological properties and were found at one time or another in our water, drinking Edmonton water is at least as safe as any other normal daily activity and much safer than most.

Where uncertainty begins to arise is with organic substances which:

1.can be found but which have not been adequately subjected to toxicological evaluation, and/or

2.the majority of organic matter which is present in drinking water which we cannot currently characterize, let alone subject to meaningful toxicology testing.

For these unknowns, we must look for evidence of harm in ways other than deductive reasoning based upon known toxicological properties. When we find no evidence in support of an adverse health effect we may not be able to answer the question about safety. However, we can answer the "concern" question by indicating that an individual's intellectual capacity for considering health questions would be better directed at more likely health risks. That response is not intended to relieve our health authorities from a responsibility to continually improve our understanding of possible health effects of trace substances. Likewise, our engineers should be optimizing the water treatment processes to eliminate unnecessary organics thereby avoiding unnecessary health evaluations.

One area which did raise concern with Dr. Bull is the potential for toxic effects from the disinfectant, chlorine dioxide or its inorganic byproduct , chlorite, if not adequately controlled. This oxidant chemical has several advantages over others available. These factors contributed to its adoption by Edmonton. Many other places in the world use it, including some of the advanced European plants. Health problems are only likely to arise if it is used without adequate care in controlling dosage. Thus, we do not expect that using this chemical at carefully controlled, low dosages will contribute any risk to consumers. But the use of this disinfectant carries with it a responsibility to employ sophisticated monitoring and dosage control.

Finally, health concerns could also arise regarding waterborne disease. Evaluation of the safety of Edmonton water in this regard provided no indication of any likelihood for transmission of a life-threatening disease. However, some concerns were expressed about the possibility of transmission of enteric diseases ranging from mild stomach upsets to severe illness in the case of *Giardia*. There was no evidence to demonstrate that the current treated water supply was contaminated by any enteric pathogens, but the experts were concerned about the potential challenges to water treatment which could arise from the contaminated raw water source at Rossdale. Consequently, they have offered a variety of recommendations on monitoring and disinfection practice to assure a high level of protection against waterborne dissease. The most important action in this regard will be relocation of the Rossdale intake to a location which will obtain raw water with quality equivalent to that available at E.L.Smith.

Dealing with question 3, several experts offered input on the question of the feasibility of dealing with the annual taste and odour problems. The common view was that there had been inadequate documentation of past odor events to allow sure-fire solutions to be proposed. However, there was equal agreement, that with proper characterization, the problem should be controllable. The first step will have to be accurately defining the specific causes, sources and distribution of the problem. Some encouraging initial steps have been made toward that goal.

Dealing with the many complex water quality issues which arise for Edmonton's water treatment plants requires expertise in several disciplines. There is scope for an in-house water quality specialist who could provide ongoing expertise for dealing with many of the complex issues.

Finally, an important coincident benefit of all of the expert visits has been the recognition that there is considerable scope for process improvement and control with the existing treatment processes. A coordinated and concerted effort to exploit those opportunities can be expected to provide savings in chemical consumption costs and significant improvements in treatment performance. The latter may reduce some of the pressures for multiple chemical disinfection barriers against microbial pathogens.

12. RECOMMENDATIONS

1. The Rossdale raw water intake must be relocated to a point upstream where it can be reliably assured of obtaining raw water quality equivalent to that now obtained at the E.L. Smith plant. Work on planning the engineering studies to verify the optimum upstream location should proceed as expeditiously as possible.

2. Edmonton Water and Sanitation should assemble a process improvement team, free from routine operational responsibilities, to initiate a series of process development goals. The team should ideally include, at least for the first 2 years while local experience is developed, a senior process development engineer or chemist with extensive experience in water process evaluation. Other members of the team should include an engineer experienced with the Edmonton water treatment plants, a chemist with good understanding of water chemistry and analysis and a technician with good chemical analysis skills.

3. The process improvement team should undertake to:

a. optimize the existing processes with the aim of achieving a finished water turbidity goal of 0.1 NTU.

b. optimize the process for raw water organics removal to minimize the required disinfectant doses

c. evaluate the benefits arising from separation of processes, particularly separation of chemical additions to the raw water.

4. Edmonton Water and Sanitation should, upon successful demonstation of the process improvement team's capabilities to assist in process upgrading, undertake to build or acquire a small scale pilot plant facility which can be used to pilot more significant future changes to the processes.

5. Edmonton Water and Sanitation should undertake to upgrade the chemical analysis capability of its water laboratory by:

a. institution of a rigorous quality assurance / quality control program concerning all analyses including those sent to outside laboratories

b. instituting a laboratory information management system to expedite data collection handling and recording.

c. devoting the resources of the equivalent of 1 full time laboratory scientist to analytical methods development.

d. arranging for a long term (minimum of 1 year), assured performance contract for GC/MS support with an outside laboratory.

6. Edmonton Water and Sanitation should undertake to upgrade microbiological analysis in support of solving some of the difficult disinfection questions raised. This should involve dedication of an experienced microbiologist with a trained technician to perform more detailed microbiological monitoring

7. Edmonton Water and Sanitation should establish a technical specialist position of water quality advisor reporting directly to management without other major administrative responsibilities. The specialist should have advanced education and experience in matters of water chemistry and treatment processes and would serve to represent the interests of the Water Treatment Plants on water quality issues in dealings with Alberta Environment, the Edmonton Board of Health, other cities and agencies and the public. This position is perceived necessary to integrate the Water Treatment Branch response to the many water quality and health issues which will inevitably arise.

8. Alberta Environment, Edmonton Water and Sanitation and the Edmonton Local Board of Health should seek assistance from the Department of National Health and Welfare for the development of separate maximum acceptable concentrations for chlorine dioxide, chlorite and chlorate. This matter has already been identified as an upcoming task for the Federal / Provincial Committee on Drinking Water.

9. Alberta Environment should institute a mandatory quality assurance / quality control program for all approved water labs to participate in. This should be the first step towards formalized water laboratory certfication. Once these quality control programs have been established, Alberta Environment should incorporate mandatory quality control procedures into operating licenses.

10. The City must adopt a policy to restrict any upstream activity within city boundaries which could compromise the water quality available at the E.L.Smith intake and the new Rossdale intake.

11. Alberta Environment should adopt a policy of no further industrial discharges to the North Saskatchewan River upstream of Edmonton. The burden of proof for any request for exception to this policy should rest with the proponent.

12. The City of Edmonton and Alberta Environment should seek to improve cooperation and information exchange concerning studies about the North Saskatchewan River. Likewise, the City of Edmonton should be encouraged to refer needs and problems to Alberta Environment for research attention. The latter agency should endeavour to be responsive to Edmonton's needs.

13. The City of Edmonton and Alberta Environment should encourage the development of a federal research program to promote water treatment technology research and development.

14. In addition to the foregoing recommendations, there are several specific recommendations which arise from the specific topic areas reviewed by the study. These detailed recommendations are provided in those sections of this report to which they apply.

15. In addition to the recommendations included in this Overview Report, individual experts have offered some specific recommendations on matters which rated as a lesser priority for dealing with the major issues raised during this study. However, these additional recommendations are worthy of consideration for dealing with those particular specific issues if they should become major concerns.

REFERENCES

1. Health and Welfare Canada. 1978. Guidelines for Canadian water quality. Prepared by the Federal-Provincial working group on drinking water of the Federal-Provincial committee on environmental and occupational health, National Health and Welfare. Canadian Government Publishing Center, Supply and Services, Hull, Quebec.

2. APHA. 1985. Standard Methods for the Examination of Water and Wastewater. 16th Ed.American Public Health Association, American Water Works Association and Water Pollution Control Federation

3. Miller, G.W., R.G. Rice, C.M. Robson, R.L. Scullin, W. Kuhn,, H. Wolf, and J. K. Carswell. 1978. An assessment of ozone and chlorine dioxide technologies for treatment of municipal water supplies. U.S. Environmental Protection Agency, **EPA-600/8-78-018**, 68 pp.

4. W.H.O. 1984. Guidelines for drinking-water quality - Vol. 1. Recommendations. World Health Organization, Geneva, Switzerland.

5. U.S. EPA. 1985. Federal Register. U.S. Environmental Protection Agency. Part IV. Wednesday, November 13, 1985. **50(219)**.

6. Lippy, E.C. 1981. Waterborne disease: occurrence is on the upswing. *J. Am.Water.Works Assoc.* 73:57-62.

7. Committee Report. 1981. Waterborne disease in the United States and Canada. By the Committee on the status of waterborne diseases in the U.S. and Canada. *J. Am.Water.Works Assoc.*. 73:528-529.

8. Lippy, E.C. 1981. Waterborne disease: occurrence is on the upswing. *J. Am.Water.Works Assoc.* 73:57-62.

9. Collier, M.K., and P. Macdonald. 1983. Giardiasis in Edmonton. Edmonton Local Board of Health. Edmonton, Alberta.

10. Amoore, J.E. , Forrestor, L.J. and P. Pelosi. 1976. Specific anosmia to isobutyraldehyde: the malty primary odor. *Chem. Senses Flavor.* **2**, 17-25.

11. U.S. EPA. 1978. Federal Register. U.S. Environmental Protection Agency. Part II. Thursday, February 9, 1978. **43(28)**.

12. Stanley Associates Eng. Ltd. 1983. Rossdale water treatment plant refurbishment - Study report raw water storage lake, July 1983. Prepared for the City of Edmonton, Water and Sanitation, by Stanley Associates Engineering Ltd., Edmonton.

13. National Research Council. 1977. Drinking water and health. Prepared by the Safe Drinking Water Committee, Advisory Center on Toxicology, National Research Council. National Academy of Sciences, Washington, D.C.

14. Gore & Storrie. 1950. The City of Edmonton report on proposed extensions of the water works system. Prepared for the City Commissioners of the City of Edmonton by Gore & Storrie Consulting Engineers, Toronto.

APPENDIX A

CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

Terms of Reference

1. Solicit, by means of public announcement, written information which may be relevant to the study.

2. Evaluate the source of Edmonton's raw water supply, namely the North Saskatchewan River, in relation to:
   - a. existing water quality criteria (Alberta, Canada, U.S. and Europe).
   - b. other raw water sources for major cities in Canada, the U.S.& Europe.
   - c. any trends which can be discerned from historical data.

3. Evaluate the methods of monitoring raw and finished water quality with respect to:
   - a. physical and chemical quality measurement,
   - b. microbiological quality measurements,
   - c. emergency warning capability.

4. Evaluate the methods of treatment employed in Edmonton in relation to :
   - a. licensed requirements
   - b. Canadian practice
   - c. U.S. practice
   - d. European practice.

5. Review the quality of Edmonton drinking water as evidenced by currently available data:
   - a. in comparison to licensed requirements , including review of any breaches of established standards and guidelines for a minimum of the past 12 months,
   - b. in comparison to finished water quality for major cities, of similar size in Canada, the U.S. and Europe,
   - c. to document the relationships of such substances or microorganisms as may already have been found in the water to known health effects
   - d. to document the relationships of such substances or microorganisms as may already have been found in the water to known aesthetic (taste and odour) concerns.

6. Review the significance of Edmonton drinking water quality as a factor in the health of Edmonton residents:
   - a. by reviewing available, relevant health statistics in comparison with other Canadian cities of comparable size,
   - b. by assessing health risks for any notable factors identified in task 5.

7. Evaluate options to alleviate health or aesthetic concerns and obtain preliminary cost estimates for preferred options. Consideration will include, at a minimum:
   - a. increased upstream pollution control,
   - b. relocation of water intakes,
   - c. reservoir options,
   - d. modified treatment technology.
   - e. point-of-use treatment options.

8. To formally solicit reviews of the draft final report, following its public release, and to consider reviews provided by interested parties in preparing the final report. The review process shall include:
   - a. solicited comments from 2 independent experts not involved in the study team,
   - b. written comments submitted by interested organizations or members of the public,
   - c. presentations at a Public Review Meeting by those individuals or groups who have responded to the request for written comments (item b).

APPENDIX B

The Expert Study Team

Study Director

Dr. Hrudey, a Professor of Environmental Engineering at the University of Alberta, coordinated all aspects of the study and wrote the overview report based on individual submissions from the selected experts. He holds a Ph.D. in Public Health Engineering from the University of London (England), has 16 years of research and professional experience concerning the control of toxic pollutants and has consulted on toxic waste management for industries and governments in Alberta, British Columbia and Australia. Dr. Hrudey has organized the experts into three broad areas: chemistry, technology and health.

Chemistry.

Dr. Ronald T. Coutts, a University Professor (the highest academic rank) in the Faculty of Pharmacy and Pharmaceutical Sciences at the University of Alberta holds a Ph.D. in Organic Chemistry from the University of Glasgow and has 30 years of research and practical experience in medicinal chemistry and analytical toxicology. Dr. Coutts served as Co-chairman of the XI Commonwealth Games Drug Testing Program, as a member of the National Health Research and Development Committee on Biology/Chemistry/Toxicology, he has contributed to 17 textbooks and is currently Editor of the Journal of Pharmacological Methods among many other academic and professional accomplishments. Dr. Coutts critically evaluated the monitoring of Edmonton water for trace organic substances.

Dr. Byron Kratochvil, a Professor of Analytical Chemistry at the University of Alberta, holds a Ph.D. in Analytical Chemistry from Iowa State University and has 25 years of research and practical experience in chemical analysis and quality control including a year with the U.S. National Bureau of Standards working on methods of sampling for reliability in chemical analysis . Dr. Kratochvil has co-authored 3 widely used textbooks in his field and serves as the Analytical Chemistry Editor for the Canadian Journal of Chemistry. Dr. Kratochvil critically evaluated the monitoring of Edmonton water for inorganic substances, routine organic analyses and the overall laboratory quality control.

Technology.

Prof. Dr. Heinz Bernhardt is Technical Director of the Wahnbachtalsperrenverband (Wahn Creek Reservoir Association) in the Federal Republic of Germany. This organization supplies drinking water for the city of Bonn and surrounding communities. Prof. Bernhardt has held his present position for about 25 years, following graduation with a doctorate in chemistry from the University of Marburg. He is also a Professor at the University of Aachen. Prof. Bernhardt is recognized as a leading authority in the field of coagulation and flocculation. He developed the Wahnback process for physical-chemical phosphorus removal and has intensively researched the disturbance of coagulation caused by algal organics.

Mr. Paul D. Foley is an independent consultant following retirement after 40 years of professional practice in water treatment. He holds a degree in Chemical Engineering from the University of Toronto and he served as chairman of the Research Advisory Committee of the Ontario Ministry of Environment prior to his retirement. Mr. Foley currently serves as Vice-Chairman of the American Water Works Association Research Foundation along with project management duties for the Niagara Falls water treatment pilot project for trace contaminant removal. Mr. Foley critically reviewed Edmonton water treatment practice in relation to practice elsewhere in Canada.

Dr. Peter M. Huck, a Professor of Environmental Engineering at the University of Alberta, holds a Ph.D. in Chemical Engineering from McMaster University and has 15 years of research and professional experience in water and wastewater treatment. He is Technical Program Chairman for the 2nd National Conference on Drinking Water (April 1986, Edmonton) and is performing a major study evaluating alternate drinking water disinfectants for National Health and

Welfare and Alberta Environment. Dr. Huck drew upon a close network of European contacts to critically compare Edmonton water treatment practice with advanced European practice.

Dr. Gary S. Logsdon is Director for Microbial and Inorganic Particle Control of the Drinking Water Research Division, U.S. Environmental Protection Agency in Cincinnati. He holds a D.Sc. in Environmental Engineering from Washington University and has 24 years of research and professional experience in drinking water treatment. He serves as Editor of the Journal of Environmental Engineering (American Society of Civil Engineers) and has specialized his interests in the matters of *Giardia* ("beaver fever") and asbestos removal in water treatment. Dr. Logsdon critically evaluated the Edmonton water treatment system for the removal of *Giardia* cysts and related contaminants.

Dr. Daniel W. Smith, a Professor of Environmental Engineering at the University of Alberta, holds a Ph.D. in Environmental Health Engineering from the University of Kansas and has 19 years of research and professional experience in water and wastewater treatment. Dr. Smith has served in numerous professional capacities including Chairman of the Environmental Engineering Division of the Canadian Society for Civil Engineering and consultant to the World Health Organization on water and wastewater services in cold climates. Dr. Smith reviewed the microbial contamination of the North Saskatchewan River andthe dispersion of storm sewer discharges within the river.

Dr. Vernon L. Snoeyink, a Professor of Environmental Engineering at the University of Illinois, holds a Ph.D. in Water Resources Engineering from the University of Michigan, has 22 years of research and practical experience in water treatment and is co-author of the graduate textbook, "*Water Chemistry*". He has served in numerous professional capacities including: Chairman of the Research Division of the American Water Works Association, Member of the Safe Drinking Water Committee of the U.S. National Academy of Sciences and consultant to the World Health Organization on drinking water treatment. He is currently a technical advisor on the Niagara Falls pilot project for trace contaminant removal from drinking water. Dr. Snoeyink critically reviewed Edmonton water treatment practice in comparison with U.S. practice.

Mr. Albert R. Trussell is Director of Research and Development, Montgomery Labs Division of James M. Montgomery, Consulting Engineers, Inc. of Pasadena, California. He holds a degree in Chemistry from the University of California at San Diego, has 10 years of professional experience in advanced water treatment and was a primary contributor to the new (1985) graduate engineering textbook, "*Water Treatment: Principles and Design*". He serves as Chairman of the Taste and Odor Committee of the American Water Works Association and has consulted on several taste and odor control problems with American water utilities. Mr. Trussell reviewed the taste, odor and color problems with springtime Edmonton water in light of similar problems and solutions elsewhere.

Health.

Dr. Richard J. Bull is Associate Professor of Pharmacology / Toxicology at Washington State University. He holds a Ph.D. in Pharmacology from the University of California, San Francisco Medical Center, has 22 years of research and practical toxicology experience and acted as Chief of the Toxicological Assessment Branch and Director of the Toxicology and Microbiology Division of the U.S. Environmental Protection Agency, Health Effects Research Lab in Cincinnati. In the latter position, he directed a program of 90 staff and $10 million of research into health effects of environmental contaminants and he has served on numerous health advisory committees. Dr. Bull received a 1984 EPA Scientific Achievement Award for research documenting health concerns with products formed by chlorination of humic substances. Dr. Bull provided an overall assessment of health concerns associated with Edmonton drinking water in view of information provided by other team members, existing data and results from 1986 monitoring.

Dr. Ian C. Munro is Director of the Canadian Centre for Toxicology in Guelph. He holds a Ph.D. in Toxicology and Pharmacology from Queens University, has 24 years of research and practical experience in toxicology and risk management and is co-editor of the recent (1985) professional book series "*Toxicological Risk Assessment*". He has served on numerous national and international advisory committees including acting as Chairman of the International Committee on Hazards Associated with Dioxin in the Great Lakes and as a member of the Subcommittee on Risk Assessment of the Safe Drinking Water Committee of the U.S. National Academy of Sciences. Dr. Munro and his colleagues at the Canadian Centre for Toxicology reviewed the scientific literature for health effects of those contaminants confirmed in Edmonton drinking water by Dr. Coutt's review of previous data and 1986 monitoring.

Dr. Pierre Payment is a Professor at the Centre for Research on Virology at the Institut Armand Frappier, Universite of Quebec, in Montreal. He holds a Ph.D. in Microbiology and Immunology from the University of Montreal and has 16 years of research and practical experience in microbiology and virology. He has served in several professional capacities including acting as a member of the Canadian Public Health Association Committee on Microbiological Quality of Drinking Water and the Committee on Swimming Pool and Recreational Water Quality. Dr. Payment reviewed the circumstances surrounding the raw water supply in Edmonton and the microbiological indicator monitoring data to assess the likelihood of viral contamination of Edmonton water and the need for virological monitoring.

Dr. Robert E. Rogers is President of Toxcon Consulting Services of Sherwood Park, Alberta. He holds a Ph.D. in Toxicology from the University of Rochester and has 13 years of research and practical experience in biology and toxicology. Dr. Rogers served as Manager of the Inhalation Toxicology Program at Alberta Worker's Health, Safety and Compensation and is currently an Assistant Professor of Biology and Environmental Science at Concordia College. He has served on several advisory committees including acting as Co-chairman of the Alberta Government Risk Assessment Committee and Chairman of the Core Committee on Hydrogen Sulphide Research for the Alberta Provincial Board of Health. Dr. Rogers investigated specific health issues raised by public submissions received up to March 1, 1986.

Dr. Richard S. Tobin is Microbiology Advisor, Monitoring and Criteria Division, Health and Welfare Canada in Ottawa. He holds a Ph.D. in Biological Sciences from the University of Texas at Austin and has 19 years of research and practical experience in microbiology. Dr. Tobin has served on several advisory committees including the American Water Works Association Committee on the status of waterborne diseases in the U.S. and Canada, the U.S. Environmental Protection Agency Taskforce on Water Purifiers and the National Sanitation Foundation Joint Committee on Drinking Water Treatment Units. Dr. Tobin reviewed the microbiological monitoring procedures for Edmonton drinking water and the North Saskatchewan River, assessed the potential risk of waterborne disease with drinking water and reported on the utility of home water purifiers for Edmonton water.

Dr. Peter Toft is Chief of the Monitoring and Criteria Division, Environmental Health Directorate, Health and Welfare Canada in Ottawa. He holds a D. Phil. in Chemistry from Oxford University and has 23 years of research experience in the health sciences field. Dr. Tobin has served on numerous national and international advisory committees involving the OECD, NATO, the World Health Organization, the United Nations Environment Program, the U.S. Environmental Protection Agency and the International Standards Organization. He also serves as the Secretary of the Federal-Provincial Working Group on Drinking Water and Co-chairman of the Ontario Ministry of Environment / National Health and Welfare Working Group on the Control and Abatement of Chemical Contaminants in Drinking Water. Dr. Toft and his staff supported the study by providing relevant information from elsewhere in Canada and abroad.

Volume

2

DRAFT FOR COMMENT

A Critical Assessment of Drinking Water in Edmonton

Vol. 2 Expert Reports

Steve E. Hrudey & Associates Ltd.
Edmonton
July 1986

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Proposal for the optimisation of the Humic- and Fulvic-Acid-elimination by softening precipitation and flocculation / filtration in the E.L. Smith Water treatment plant, Edmonton.

by
Prof. Dr. Heinz Bernhardt
Siegburg, F.R.G.

Humic and fulvic acids as precursor compounds

The numerous studies into the precursor properties of natural organic substances have verified that humic and fulvic acids - just like algogenic organic matter - determine the potential of a water to yield trihalogen compounds. Should water containing such substances be chlorinated as an oxidation step within the scope of prechlorination, considerable quantities of trihalomethane compounds may arise in the drinking water depending on the concentration ratio chlorine:precursor compound. By way of example, Brett and Calverley (1) refer to chloro- and bromomethanes being able to occur in considerable concentrations in a drinking water distribution system as a function of the water temperature and the residence time of the water in the distribution system. They give humic acids in the raw water as the cause. If the formation of such organochlorine compounds in drinking water is to be prevented, the chlorine treatment must be dispensed with or the precursor compounds removed prior to chlorination by a technical scale process. This also applies to the disinfection of drinking water subsequent to treatment with chlorine.

With regard to trihalomethane compounds forming in drinking water purification Singley and coworkers (2) come to the following conclusions:

1. Precipitative softening reduced the concentrations of trihalogenated methanes produced by chlorination of groundwater and their precursors.
2. Minimum trihalomethane concentrations resulted when chlorination was practised after settling and filtration.
3. Chlorination of the raw water led to the highest concentrations of trihalomethanes.
4. Recarbonation reduced the concentrations of trihalomethanes in the finished water except when the raw water was chlorinated and an excess of chlorine

was added to the finished water followed by two days' storage to simulate retention in the distribution system.

They recommend two points, whereby with regard to the second point it is, however, to be observed that in the meantime treatment processes now exist to remove humic and fulvic acids from the raw water as essential precursors for THM formation.

1. Chlorination in precipitative softening plants be practised as late in the process as is practical, consistent with the public health protection provided by adequate retention time for disinfection to take place prior to discharge into the distribution system.
2. Emphasis on further research be directed to precursor removal. This should include studying the use of the various types of coagulant and coagulant aids as well as use of powdered activated carbon.

The mechanism of chlorine and bromomethane derivatives forming during chlorination (without direct addition of bromine, but only through the reaction of chlorine with the bromide ions present in the water) has been largely elucidated in recent research. The studies of Grandet, Weil and Quentin (3) summarized the reaction mechanisms occurring during chlorination in drinking water production as follows:

Chlorine normally reacts with the organic matter in water which has precursor properties, chloroform basically being formed. Should, however, bromide ions be present in the water at the same time, then trihalomethane compounds containing bromine will also occur. Here $CHCl_2Br$ and $CHClBr_2$ are, above all, formed. One possibility of how brominated THM compounds arise is by the bromide ions reacting with chlorine to form bromine and hypobromide. These reaction products react with the organic matter further, forming $CHCl_3$ and $CHBr_3$. However this reaction does not appear so probable as the second reaction mechanism, with chloroform as an intermediate. In this case in the presence of excess chlorine, chloroform forms an intermediary product (trichloromethyl anion), which in the presence of bromide ions reacts via intermediary steps, to form the above-mentioned bromine-containing THM compounds.

The significance of the turbidity substances for THM-formation in river water loaded by erosion products from farmed areas as a consequence of increased run-offs after periods of rain and snow melt is shown in the study of Morris and Johnson (4) using the example of the Mississipi River and the drinking water treatment of the City of Iowa. Treatment processes leading to a drastic reduction in the turbidity concentration without prior chlorination resulted in considerably lower THM concentrations than treatment processes, where the raw water containing turbidity substances was initially subjected to intense chlorination and subsequently treated by lime softening without coagulants being employed. The figures listed below indicate that the reaction leading to THM formation at an alkaline pH of 10.5, as was carried out for lime softening in one of the two water works, is intensified:

	Treatment with prechlorination and lime softening		Treatment with alum-coagulation (Al_2SO_4 coagulation) and powdered activated carbon before chlorination	
	$CHCl_3$ (µg/l)		$CHCl_3$ (µg/l)	
	16.9.75	19.9.75	16.09.75	19.9.75
Chlorination:	74	35	----	----
Softening:	148	110	----	----
Recarbonated:	128	94	----	----
Drinking water:	120	98	----	----
Drinking water:			30	28

(Data from Tab. 3 from (4))

Under alkaline conditions THM formation occurs considerably faster than under neutral to weakly acidic pH conditions. This means that the shift of the pH value into the alkaline range causes the THM forming reaction of chlorine with humic acids to increase (5). In this context, softening combined with prechlorination must be regarded as essential for THM formation, if precursors are not largely removed from the water prior to chlorination, or prechlorination as an oxidation step is not performed. Stevens and coworkers (5), for example, make reference to this on the basis of numerous investigations.

The trihalomethane compounds formed can be removed from the water, at least in part, by post filtration with granulated activated carbon (GAC). However, this does presuppose that the adsorption efficiency of the granulated active carbon is not consumed by other, likewise adsorbable organic substances. Compared to other adsorbable organic substances trihalomethane compounds are not easily adsorbed and more easily desorbed.

The operation time of granulated activated carbon filters (GAC) is relatively short for adsorption of trihalomethane compounds necessitating frequent regeneration of the granulated activated carbon filters.

The use of powdered activated carbon gives better results, although a sufficiently long contact time of the activated carbon with the water (at least 15 minutes) is necessary and the water not contain any substances which may affect the adsorption efficiency of the powdered activated carbon.

That suspended materials can be decisive for THM formation in a water can also be seen from the fundamental studies conducted by Stevens and coworkers (5). These studies were carried out in 1976 in connection with preventing trihalomethanes from forming when using chlorine in drinking water treatment. In this case, organic matter was involved such as humic and fulvic acids and other polymeric anions which are present in surface waters as natural products of biological processes. Rook (6) referred to the significance of humic substances as precursors for THM formation during chlorination of water as part of the treatment process.

The studies in this field that have been conducted in international spheres in the last ten years have shown that such organic substances effective as precursors can be fixed to a considerable degree to the surface of suspended particles by adsorption.

A great reduction in the potential of trihalomethanes forming in a surface water is, thus, attained by removing particulate matter prior to chlorination.

Drawbacks of prechlorination with regard to THM formation

Based on the studies of the drinking water in 18 towns, the survey of Singley and coworkers (2) shows that particularly increased THM concentrations were found, when precipitative softening was carried out by adding lime water at pH > 9 after chlorination.

These results, however, should not allow the conclusion to be drawn that THM formation is increased by a softening plant. On the contrary it is possible for the precursor concentration to be reduced considerably by a softening step at the start of treatment. It only has to be ensured that the raw water is not first chlorinate

The investigations of Singley and coworkers (2) on the Daytona Beach Water Treatment Plant can be taken as an example. In this plant lime is added to the raw water in a primary mixing zone with an anionic polyelectrolyte. After 90 minutes of sedimentation the water is fluorinated and passed to a recarbonation tank, where carbon dioxide is added. Thereafter, 2-layer filtration is carried out.

The raw groundwater in this case is coloured due to humic sustances from the marshy subsoil of the catchment area of the wells.

It is evident from the description of the plant that approx. 8 mg/l chlorine is added to the raw water (prechlorination). After the softening plant, another 10 mg/l is added and after filtration another 4 mg/l Cl_2. For softening, approx. 220 mg/l of lime is added. The result is a pH value of approx. 9.5. Depending on the addition of CO_2, the pH value of the drinking water is between 7.4 and 8.2. The calcium content amounts to approx. 20 mg/l, total hardness 85 mg/l as calcium carbonate and the turbidity 0.3 FTU.

Subsequent to prechlorination, the THM concentration in the raw water amounted to 3.02 µmol/l TTHM and 328 µg/l chloroform. The chloroform concentration in the treated water can be changed according to when the chlorine is added to the treatment.

The essential result of these studies is that the softening process is effective with regard to the removal of the precursors. This indicates that precipitation of calcium carbonate with adsorption of the precursors on the precipitation products and subsequent removal of the precipitation products leads to an effective reduction of these substances and that trihalomethane compounds are formed to a lesser degree in the following chlorination step.

Moreover, recarbonation results in THM formation being reduced, probably due to the fact that the pH value is returned to the neutral range. This also shows that, where possible, chlorination for disinfection should be carried out at the end of treatment when the pH is no longer strongly basic.

On the whole, the results of this study indicate that concentrations of trihalomethane compounds can be reduced by more than 50 % by a softening process, if there is no prechlorination; chlorination should only occur at the end of treatment exclusively for disinfection purposes, whereby it is desirable that alkaline conditions are no longer present.

Technical process proposal for removing humic and fulvic acids

Removing natural organic macromolecular compounds, such as humic and fulvic acids as well as algogenic organic matter, can be optimized, if the two mechanisms are implemented for removing these substances consecutively:

a) Precipitation and coprecipitation of humic and fulvic acids as calcium humate and calcium fulvinate together with precipitation of calcium carbonate at pH 10.5 with subsequent separation by sedimentation in the sludge contact process.

b) Flocculation with aluminium salts and formation of mixed ligand complexes between trivalent metal hydroxo complexes and oxide hydroxides with humic and fulvic acids at pH 6 to 6.5 with subsequent removal by filtration (7.8).

Studies by Haberer and Normann (9) have shown that removal of such organic substances by flocculation is very much dependent on the pH value (Fig. 1). The worst removal rate is with a pH value of 8, the best removal being with pH 5 and pH 11.

Resultant from this is a two-stage process for removing humic and fulvic acids, which has been utilized on a technical scale. The results under operation conditions on a technical scale can be seen in Table 1. In these time-limited, technical experiments the dose of ferric salt was not optimized for humic acid removal. The flocculation pH value of 7.4 was also too high. Moreover, these experiments were carried out without sludge contact. Removal of humic acids could have been increased more with sludge contact ($CaCO_3$).

Such optimized conditions were implemented on a laboratory scale and are summarized in Table 2.

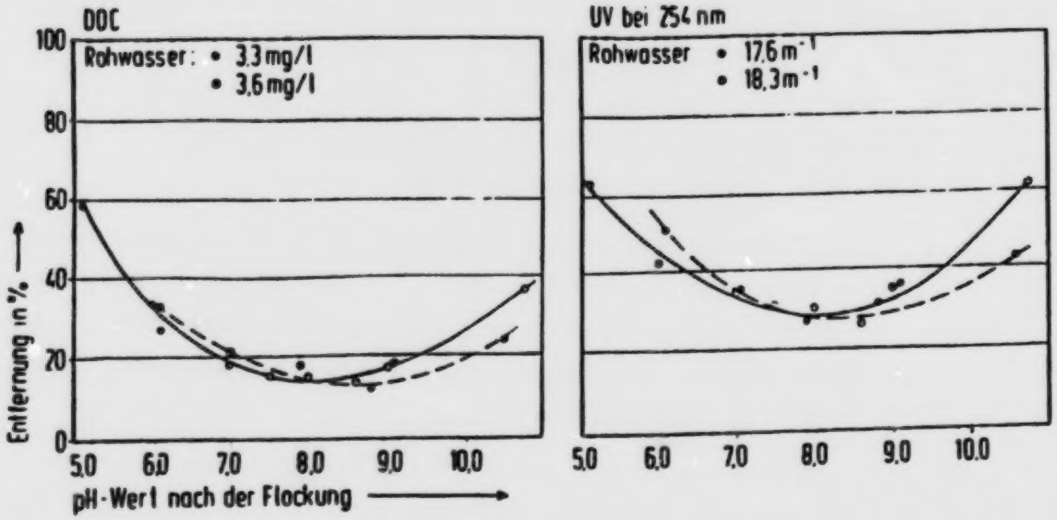

Fig. 1 pH dependence of the removal of organic matter with Fe(III)-flocculation, investigated on two different Rhine waters with $FeCl_3$ additives: ● 0.09 mol/m^3 and ○ 0.12 mol/m^3. (From (9))

Table 1: Efficiency of two-stage precipitation without sludge contact on a technical scale (from (9))

Process step :	Raw water		Dosage		After the reaction			
	DOC g/m³	UV m^{-1}	$Ca(OH)_2$ in g/m³	$FeCl_3$ in g/m³	pH	m-value	% DOC Removal	%UV
pre-treatment	5.2	10.5	100	./.	10.2	2.0	8-21	-
with $Ca(OH)_2$, 10°C	(3.6...6.2)	(9.5..10.5)						
consecutive	4.4	9.8	./.	30-45	7.4	2.0	40	51
Fe(III) flocculation	(4.2...6.0)	(8.5...12.5)						
Total process							44	61
pretreatment with	4.1	12.0	82	./.	10.7	1.0	17	-
$Ca(OH)_2$, 10°C	(3.4...4.8)	(10...13)						
consecutive	3.4	7.7	./.	33-38	5.9	1.0	46	66
Fe(III) flocculation	(3.1...4.0)	(7.0...9.0)						
Total process							58	70

Table 2: Removal of organic matter from Rhine water under various flocculation conditions, expressed in % of DOC (laboratory experiments)

	high mol. substances	high mol. acids	humic acid
Rhine water DOC in g/m^3	3.4	11.08	0.27
% of DOC	67	21	5
Efficiency of treatment			
1. Flocculation with $Ca(OH)_2$ with pH 10.5	35	63	89
2. Flocculation with $FeCl_3$ with pH 6.1	25	72	100
3. 2-stage flocculation a) $Ca(OH)_2$+$CaCo_3$ b) $FeCl_3$ with pH 6.1	77	99	100

Combined process, point 3:

First step:

Flocculation, precipitation and coprecipitation by adding 135 g/m^3 $Ca(OH)_2$ at pH 10.5. Removal attains the highest values, when this step is carried out in a sludge contact process. When the content of bicarbonate ions in the water is high enough, the calcium carbonate flocculation arising in this process step is adequate to attain the good removal efficiency. However, what is decisive here is the contact with the precipitated calcium carbonate in the course of the sludge contact process.

Second step:

Flocculation with 45 mg/l $FeCl_3$ at pH 6.1. The acidic pH must be achieved by addition of acid. This can occur either by adding CO_2 within the process of recarbonation or, should this not be sufficient, by adding small quantities of acid with the flocculant. The previous entcarbonization of the water causes the buffer capacity of the water to be reduced to such a degree that no large quantities of acid have to be additionally dosed to achieve this pH value.

Third step:
Subsequent filtration ensures that all the precipitates and flocs are adequately removed from the water both from the first as well as the second step.

The objective of treatment is turbidity of the filtrate of $\leqq$ 0.1 TU/F. This can be attained in this manner.

With the aid of this combination high molecular weight, organic acids can be removed practically completely and the total contents of high molecular weight organic matter by about 80 %.

However, this process run on a technical scale will result in lower removal values at water temperatures below 10°C. To what extent the removal capacity drops due to this must be investigated in preliminary experiments in a pilot plant. The removal capacity must not, however, be below 50 %. It is however possible - particularly at low water temperatures - to increase insufficient removal by adding flocculant in two consecutive dosages.

Depending on the flocculant used, the optimum pH value has to be produced in order to remove humic and fulvic acids. According to studies conducted by Sontheimer (10) the pH value for aluminium sulfate (dosage: 3 mg/l Al) is 5.5 - 6.5, whereby optimum removal of the fulvic acids occurs with pH 5.5, optimum removal of the particulate matter including the aluminium oxide hydroxide flocs is, however, pH 6.5. Therefore, when aluminium sulfate is used as flocculant the pH must be adjusted to the optimum value of 6.5. Should polyaluminium chloride be used as a flocculant instead of aluminium sulfate, the optimum flocculation pH value is likewise 5.5 - 6 and optimum particle removal, in turn, pH 6.5. Reduction of turbidity is, however, largely independent of the pH value.

On the other hand, ferric chloride is not so well suited for removing humic and fulvic acids. Absolute removal efficiency for fulvic acid, which is achieved at pH 4 - 4.5, is somewhat lower than the removal efficiency which can be achieved by using aluminium salts. Turbidity reduction and the lowest residual amount of flocculant in the filtrate is achieved with pH 5. Due to the fact that implementation of a flocculant pH value between 4 and 5 can hardly be technically realized, but that fulvic acid removal at pH 6.5 only amounts to about 40%, not iron, but aluminium salts should be used as flocculant. The optimum pH value for flocculation must be determined by prestudies in a pilot plant.

The dose of flocculant depends on the content of organic matter in the water to be flocculated. Studies by Jekel (11) show that the removal of natural organic matter (expressed as DOC) is optimum, when the dosage amounts to approx. 0.7 g Al/g DOC (see Fig. 2). Prestudies on a pilot scale are likewise necessary to determine the optimum aluminium salt dosage.

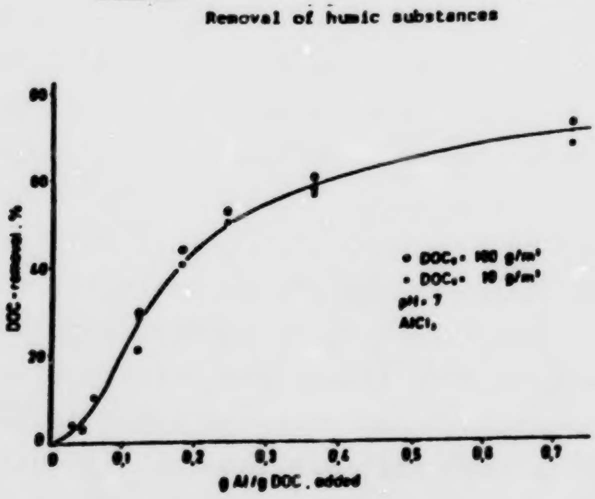

Fig. 2: Removal of dissolved organic carbon (DOC) by coagulation of a fulvic acid at neutral pH (from (11))

Humic and fulvic acid removal in the E. L. Smith Water Treatment Plant, Edmonton

This two-stage precipitation can now be applied to the E. L. Smith Water Treatment Plant without any great modifications, whereby as the first step, flocculation needs to be carried out in the strongly basic pH range. This is recommendable, because the pH value of the raw water amounts to ~ 8.2. Under these conditions, really good remov rates are achieved for dissolved organic matter. In this manner, DOC removal in February 1986 for a raw water containing 22 mg/l DOC amounted to 75%. About 6 mg/l DOC was measured in the filtrate. This figure is, however, still too high, if formation of THM is largely to be avoided in the water when a subsequent chlorine disinfection is carried out. Further DOC reduction does appear necessary due to the fact that the WHO has published (12) a Guide Value of 30 µg/l for chloroform.

In February 1986 the concentration of humic acid in raw water was between 5 and 6 mg/l and in finished water between 3 and 4 mg/l. This indicates that with lower concentrations of humic acid in the raw water and at low water temperatures the elimination capacity of the E. L. Smith Plant reduces to 40 - 50 %.

Formation of trihalomethane compounds can be reduced by using chlorine dioxide in addition to chlorine. Nonetheless it must not be overlooked that chlorine dioxide is at least partly reduced by natural organic matter with chlorite being formed. In the Federal Republic of Germany the amount of chlorite in drinking water must not exceed 0.2 mg/l. Thus, limits are set to chlorine dioxide being added. In our experience reduction of chlorine dioxide with such organic matter is slow and we have found that 80% of the chlorine dioxide is reduced to chlorite with the water having a retention time of 100 hours in the distribution system (13).

If the actual effects of the chlorine dioxide dose are to be determined, the analyses have to be carried out after a period of 100 hours. Knowing this, the chlorite concentrations which are actually in the drinking water can be determined. The same applies for trihalomethanes. Formation of these organic compounds is time dependent. Decisive is the concentration of trihalomethane compounds at the point of utilization. For this reason, determination of the concentration of trihalomethanes in water after chlorination should also be carried out after a period of 100 hours.

For optimizing the plant the following treatment scheme is proposed:

1. The chlorine dioxide dose at the commencement of the treatment process should be omitted. This will result in a substantial decrease in the reduction of chlorine dioxide by the organic matter present in the raw water, which is removed from the water in the course of the treatment process.

2. Lime water should be added in mixer 2 and a pH value of 10 - 10.5 achieved.

3. In clarifier 1 an initial softening process, an elimination of humic and fulvic acids under alkaline conditions and initial reduction of the turbidity content occurs. The clarifier is to be operated with sludge contact in order to utilize the adsorbing effect of the precipitated $CaCO_3$.

4. In mixer 2, sodium hydrogen carbonate and again lime water should be added when required in order to continue the softening process and the elimination of organic matter in the clarifier 2. This is particularly advantageous at low water temperatures.

After the second clarifier, the stage of alkaline precipitation is completed.

5. In the recarbonation chamber 1, the addition of CO_2 is provided in the scheme. Depending on the buffer capacity of the water, sulfuric acid should be added to achieve a pH value of 6 - 6.5.

6. Following adjustment of the pH value, aluminium sulfate or polyaluminium chloride is dosed in mixer 3. The amount of flocculant to be added as well as the type of flocculant (aluminium sulfate or polyaluminium chloride) must be determined in pre-studies. Where necessary, a polyelectrolyte is also to be added here in order to improve removal values for particulate matter and humic substances.

7. Contact flocculation occurs with the added aluminium salt in clarifier 3. During this process the humic and fulvic acids remaining in the water are adsorbed by mixed ligand complexes forming on the aluminium oxide hydroxide flocs. The main removal of flocs and turbidity occurs by sedimentation in clarifier 3.

8. Instead of recarbonation in chamber 2, aluminium sulfate or polyaluminium chloride is again added. Here, too, provision should be made for adding a polyelectrolyte to produce flocs, which can be optimally removed by filtration. The recarbonation step in chamber 2 should be modified, so that the flocculation chemicals to be added are mixed with the water adequately.

9. Filtration takes place directly afterwards. The repeated dose of flocculant results in the particles carried out of clarifier 3, which are difficult to remove by filtration, being converted to a form which can be removed well in the subsequent filtration.

10. The object of treatment must be:

DOC content	<	1 mg/l
Al content	<	20 µg/l
TU/F	<	0.1

11. Subsequent to filtration, a pH value in the finished water must be achieved by renewed addition of lime water to ensure that the water is not aggressive. The pH adjustment must be determined by measuring the aggressiveness of the finished water. Instead of lime water, caustic soda can also be used.

12. Following filtration, the water must be disinfected by adding chlorine and chlorine dioxide. The combined dosage of chlorine and chlorine dioxide has the advantage of assuring adequate disinfection. On the other hand, the subsequent dosage of chlorine dioxide causes a possible haloform reaction of the chlorine with the organic matter remaining in the water to be prevented. This combination of chlorine with subsequent dosage of chlorine dioxide was developed by the City of Zürich Water Company and has proven itself there for reducing the formation of trihalomethanes (14, 15).

Rossdale Water Treatment Plant

The treatment system of the Rossdale Water Treatment Plant was to be modified to function in the same way as the E. L. Smith Water Treatment Plant. The most important measure to be carried out immediately is to locate the raw water source of the Rossdale Water Treatment Plant to the area of the E. L. Smith Water Treatment Plant. This can be achieved by laying a pipeline in the Saskatchewan River. This ensures that the Edmonton street run-off, which flows into the

Saskatchewan River above the Rossdale Water Treatment Plant, does not get into the withdrawal region of the Rossdale Water Treatment Plant.

It cannot be ensured - no matter how complicated the treatment technology - that the substances getting into the river via the stormwater drains are eliminated to the extent that is necessary for securing the drinking water supply of a city like Edmonton.

Only this measure allows the drinking water supply of Edmonton to be secured for the whole year. Otherwise, due to the unforeseeable worsening of the quality of the left side of the river as a result of the discharge of rainwater it is not possible to achieve a drinking water with the required standard despite extensive treatment measures. Moreover, this technical measure is considerably less expensive to operate than investing in and running a water treatment plant equipped with all the requisite paraphernalia.

lterature

1.) R.W. Brett and R.A. Calverley: A one year survey of changes in Trihalomethane concentrations within a distribution system. J. AWWA (1979), S. 515 - 52o.

2.) J.M. Singley and coworkers: Minimizing Trihalomethane formation in a softening Plant. Municipal Environmental Research Laboratories, Office of Research and Development, US EPA-Report.

3.) M. Grandet, L. Weil, K.-E. Quentin: Bildung bromhaltiger Trihalogenmethane im Wasser, ihre Verhinderung und Möglichkeiten der Elimination. Z. Wasser-Abwasser-Forsch. 19, (1986), S. 66 - 71.

4.) R.L. Morris, L.G. Johnson: Agricultural runoff as a source of halomethanes in drinking water. J. AWWA 68 (1976), S. 492-494.

5.) A.A. Stevens, C.J. Slocum, D.R. Seeger, G.G. Robeck: Chlorination of organics in drinking water. J. AWWA 86 (1976), S. 62o - 635.

6.) J.J. Rook: Formation of Haloforms during Chlorination of natural waters. Water Treatment and Examination 28, Part 2, S. 234

7.) H. Bernhardt und Mitarb. Allgemeine Gesichtspunkte bei der Verfahrenswahl für Flockung und Flockenabtrennung. DVGW-Schriftenreihe Wasser 42, (1985), S. 321 - 364

8.) N. Narkis and M. Rebhuhn: The mechanism of flocculation processes in the presence of humic substances. J.AWWA 67 (1975), S. 1o1 - 1o8

N. Narkis and M. Rebhuhn: Stoichiometric Relationsship between humic and fulvic acids and flocculants. J. AWWA 69 (1977) S. 325 - 328

9.) K. Haberer und S. Normann: Entfernbarkeit organischer Stoffe aus Rheinwasser durch Fällung und Flockung. VOM WASSER 47 (1976), S. 399 - 419

1o.) H. Sontheimer: Flocculation in water treatment. In: K.J. Ives (ed). The Scientific basis of flocculation Sigthoff and Noordhoff, Alphen aan den Rijn, Stevenson, F.J., Humus Chemistry; John Wiley and Sons, New York (1982).

11.) M. Jekel: Die Komplexierung von Aluminiumionen mit Huminsäure im Flockungsprozeß. VOM WASSER 61 (1983), S. 349 - 361.

12.) WHO: Guidelines for Drinkingwater Quality, Vol. 1: Recommendations, WHO Genf (1984)

13.) H. Bernhardt, A. Wilhelms, E. Klein: Erfahrungen mit der Desinfektion von Trinkwasser durch Chlordioxid. 38. Internationale CEBEDEAU-Konferenz, Brüssel (1985), S. 193 - 231

14.) M. Schalekamp: Control of organic chlorine compounds by pre- and intermediate oxidation.Second National Conference on Drinking Water "Treatment for Organic Contaminants" Edmonton (1986)

15) J. Valenta: Chlorite et chlorate en tant que sous-produits de réaction résultant du traitement des eaux par le bioxyde de chlore et leur surveillance analytique. International Workshop on Water Disinfection, Mulhouse (1986)

Analysis of taste and odour compounds in the Edmonton water supply, March, 1986.

Preliminary Report

May, 1986

PRE-PUBLICATION COPY
RESTRICTED DISTRIBUTION

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MANAGEMENT PERSPECTIVE

The North Saskatchewan river is the water supply for the City of Edmonton. The City has two water treatment plants which serve a total population of 700,000. Episodes of objectionable taste and odour occur regularly in the spring. Until recently, the approach has been to treat the symptom rather than understand the cause. Partly in response to public concern over the quality of their drinking water, the City commissioned Prof. Steve Hrudey at the University of Alberta to design a detailed study of drinking water quality, including taste and odour.

In early March, 1986, during a period of unusually warm weather, the city water supply developed an odour caused by upstream runoff. The University of Alberta provided several samples to analyze for geosmin and 2-methylisoborneol. We have been studying taste and odour problems caused by these two naturally-occurring substances, and have specialized in analyzing and identifying them. This report contains the results of our analysis of the Edmonton samples. Geosmin was tentatively identified in several of the samples near the threshold odour concentration. The presence of 2-methylisoborneol could not be confirmed or ruled out.

Abstract

During early March, 1986, the Edmonton water supply developed an objectionable odour due to upstream runoff. This odour was subjectively described as "grassy" or "camphor-like," suggesting that it was caused by geosmin or 2-methylisoborneol. The University of Alberta provided us with samples to analyze for these two compounds. Geosmin was tentatively identified in several of these samples. Samples of raw and finished water collected March 7 at the E. L. Smith and Rossdale treatment plants contained 0.01-0.03 µg/L of geosmin, similar to the threshold odour concentration of 0.02 µg/L for geosmin. The presence of 2-methylisoborneol was suspected on the basis of the "camphor-like" odour reported in the water and observed in the hexane extracts. However, we were unable to confirm that 2-methylisoborneol was in the samples. The hexane extracts had complex odours, suggesting that a variety of odiferous compounds was present in the water. Attempts to identify these substances by gas chromatography-mass spectrometry were unsuccessful.

Introduction

Taste and odour episodes in the Edmonton water supply are a common problem in the spring (Walker et al 1986). A comprehensive study of this problem is being carried out for the City of Edmonton by Prof. Steve Hrudey at the University of Alberta. During March, 1986, the city water supply had a strong odour subjectively described as "camphor-like" or "grassy." This suggested the presence of 2-methylisoborneol (MIB) and/or geosmin in the water. These are naturally-occurring, moderately volatile, organic compounds with threshold odour concentrations (TOC) of 0.01-0.02 µg/L. We have been studying the occurrence of these compounds in Canadian water supplies and the taste and odour problems which result from their presence. The situation in Edmonton is of special interest because of the large population affected and because the problem occurs in late winter/early spring. Geosmin production is usually associated with mid-summer blooms of blue-green algae in lakes. Because of our interest in this subject, we welcomed the opportunity to work with Prof. Hrudey on this problem.

Raw and finished water samples (2 L) collected March 7 and 8 at the E. L. Smith and Rossdale water treatment plants were sent to us for analysis. The results indicated low levels of geosmin in these samples, but the concentrations were too low to attempt confirmation by gas chromatography-mass spectrometry (gc-ms).

In addition, the odours were complex indicating that a variety of odour-causing compounds was present in the water. For further work on identification of compounds, a larger sample (16 L) of Rossdale raw water (March 4) was processed.

Methods

Samples were shipped from Edmonton by air and kept cool until processing. Just before extraction, they were warmed to room temperature and the odour noted (Table 1). The 2 L samples were extracted by stirring with hexane in a volumetric flask (Brownlee et al. 1984). The extracts were dried, an internal standard (octadecane) added, and the extracts concentrated to 0.1-0.2 mL under a stream of argon using isooctane as the "keeper" solvent. The extracts were then analyzed by gas chromatography on a 30 m DB-17 fused silica capillary column programmed from 80-260 C at 4 deg/min in a Hewlett-Packard 5710 chromatograph using a flame ionization detector. Peak areas were measured with a Hewlett-Packard 3392 integrator. The odour of the hexane-extractable material was determined by evaporating a 1-2 μL aliquot on a tissue. The odour descriptions and quantitative results are in Table 1.

The large volume sample was extracted in 2 L portions by the same method and the extracts were combined. These were used for qualitative work only. The retention time of geosmin was compared with authentic material on three different columns (Table 2) by co-injecting sample and standard. This sample was also analyzed by gc-ms in the Water Quality National Laboratory, Burlington.

Results

The subjective odour evaluations for the March 7 and 8 samples from both water treatment plants are given in Table 1. The water samples did not have strong or distinctive odours. However, the hexane extracts had strong and complex odours such as swampy, camphor, mildew, or hay. Gas chromatography of these extracts showed numerous small peaks with a few larger ones (Fig. 1). Several chromatograms contained peaks with retention times corresponding to geosmin. The detection limit was about 0.01 μg/L. Measurable concentrations of 0.01-0.03 μg/L were found in the March 7 samples and one March 8 sample (Table 1). These concentrations are near the threshold odour concentration of about 0.02 μg/L, so at least some of the odour could have been due to geosmin.

Several extracts had a camphor odour and the chromatograms had a peak with retention time very similar to, but not identical with, that of MIB. On the basis of this, we can only say that MIB might have been present in the samples. If this peak was due to

MIB, then the concentrations were similar to those found for geosmin.

After these results were obtained, it was clear that a larger sample was needed for qualitative analysis for two reasons: 1) to (attempt to) confirm the identification of geosmin and MIB, and 2) to try to identify other compounds responsible for some of the other odours. The large sample of Rossdale raw water from March 4 was analyzed by gc-ms in the Water Quality National Laboratory. Both full-scan and multiple ion detection (MID) methods were used. The results were not conclusive. MIB could not be confirmed by either method. MID showed ions for the base peak (m/z 112) and molecular ion (m/z 182) with the correct retention time for geosmin. Full scan runs failed to produce a clean spectrum for geosmin due to the complex background in the sample. However, a spectrum was obtained at the expected retention time which had ions at m/z 112 and 182.

Because we were unable to obtain a clean spectrum for geosmin, we resorted to retention time comparison by gc using columns of differing polarity. Three columns were used: non-polar (DB-1), moderately polar (DB-17), and polar (Supelcowax 10). The results are shown in Table 2 and Figs. 2 to 4. In all three cases, the peak assigned to geosmin was enhanced by co-injection with authentic standard. A peak with retention time corresponding to MIB was present in each case. These results and the gc-ms results can be explained in two ways: 1) MIB is present, but the concentrations are too low to be detected by gc-ms or it is masked by closely eluting compounds; 2) a compound with properties very similar to MIB is present.

Summary

Geosmin has been tentatively, but not positively, identified in raw and finished water samples from the E. L. Smith and Rossdale water treatment plants. Concentrations on March 7, 1986, were near the threshold odour concentration. From the complex odours observed in hexane extracts of these water samples, it is apparent that the odour is not due to geosmin alone, but to a variety of substances. The presence of 2-methylisoborneol in these samples could neither be confirmed nor ruled out.

Acknowledgements

I would like to thank John Carron of the Water Quality National Laboratory for doing the gas chromatography-mass spectrometry on these samples. The geosmin standard was provided by the Drinking Water Research Division of the United States Environmental Protection Agency, Cincinnati, Ohio.

References

Brownlee, B. G., D. S. Painter, and R. J. Boone. 1984. Identification of taste and odour compounds from western Lake Ontario. Water Poll. Res J. Canada 19: 111-118.

Walker, G. S., F. P. Lee, and E. M. Aieta. 1986. Chlorine dioxide for taste and odor control. Jour.AWWA 78(3): 84-93.

Table 1. Results for E. L. Smith (ELS) and Rossdale (RD) raw water (RW) and finished water (FW) collected March 7 and 8, 1986.

Sample	Water Odour	Hexane Extract Odour	Geosmin Conc. µg/L
Mar. 7			
ELS-RW	faint earthy	swampy camphor	0.01
ELS-FW	nearly odourless	camphor	0.02
RD-RW	nearly odourless	mildew, hay camphor	0.03
RD-FW	chlorinous	complex	0.03
Mar. 8			
ELS-RW	sulfurous	swampy	ND
ELS-FW	chlorinous	mildew	ND
RD-RW	faint earthy	swampy mildew	ND
RD-FW	weak chlorinous	swampy camphor geosmin	0.01

ND Not detected; below the detection limit of about 0.01 µg/L.

Gas chromatography conditions: 30 m by 0.25 mm id DB-17 column programmed from 80-260 C at 4 deg/min; He carrier at 0.8 mL/min; injector 250 C; flame ionization detector 300 C.

Table 2. Retention time comparisons on three columns for the hexane extract of March 4 Rossdale raw water against MIB and geosmin standards.

Column	"MIB" Ret. Time, min Sample	Sample+Std	"Geosmin" Ret. Time, min Sample	Sample+Std
DB-1	9.27	9.28	15.82	15.80
DB-17	10.72	10.76	18.48	18.49
Supelcowax 10	12.90	12.88 *	19.30	19.30

Sample+Std runs were co-injections of sample and standard.

* This retention time is for the standard only.

Gas chromatography conditions: columns were fused silica capillary columns 30 m by 0.25 mm id; He carrier flow 0.8 mL/min; initial temperature 80 C; final temperature 260 C for DB-1 and DB-17, 240 C for Supelcowax 10; temperature program rate 4 deg/min; injector temperature 250 C; flame ionization detector temperature 300 C for DB-1 and DB-17, 250 C for Supelcowax 10; instrument Hewlett-Packard 5710 chromatograph with 3392 integrator.

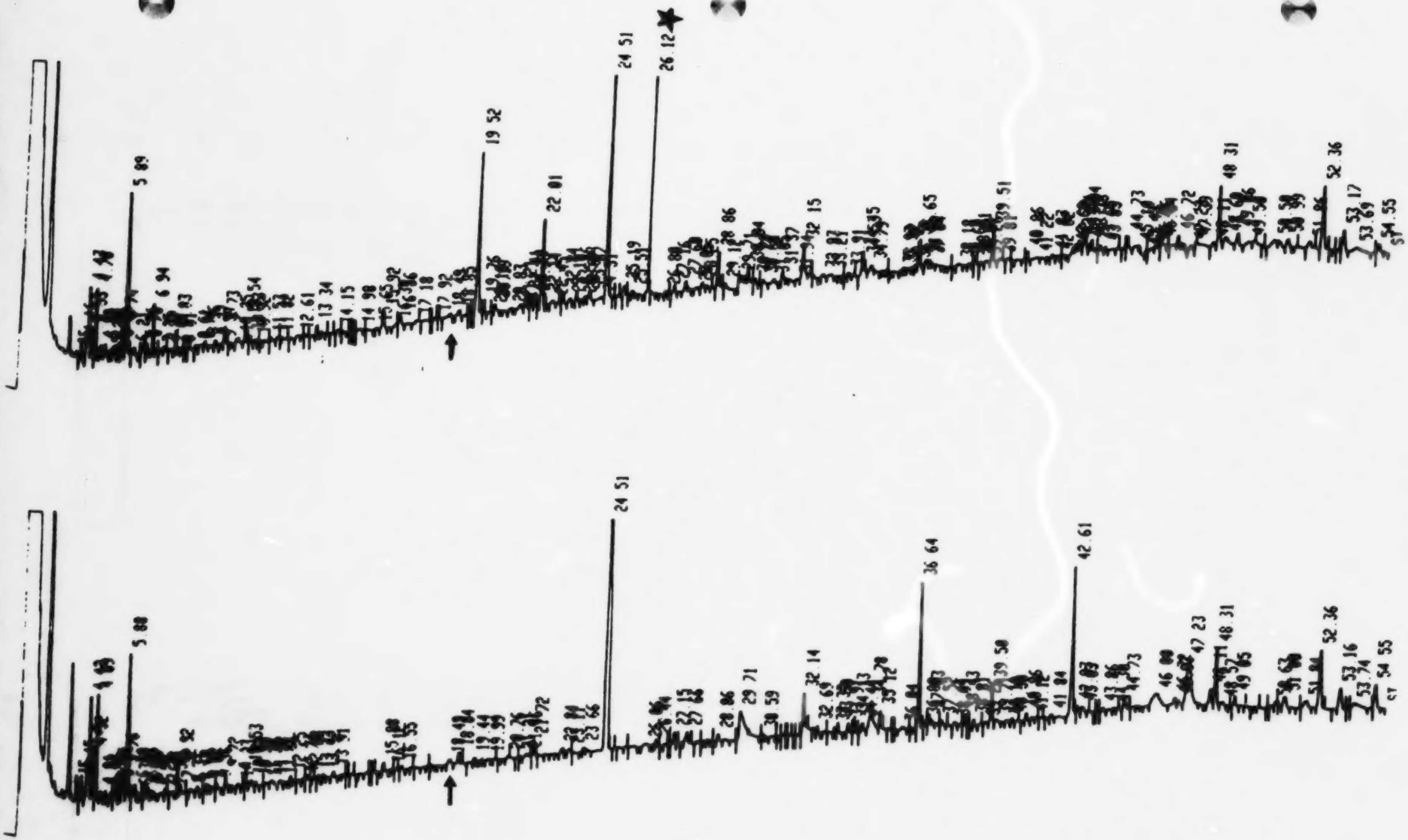


Fig. 1. Gas chromatograms of hexane extracts of E. L. Smith finished water (top) and Rossdale finished water (bottom) collected March 7, 1986, and run on a 30 m DB-17 column. The geosmin peaks are noted with an arrow (↑), and noise spikes with a star (★).

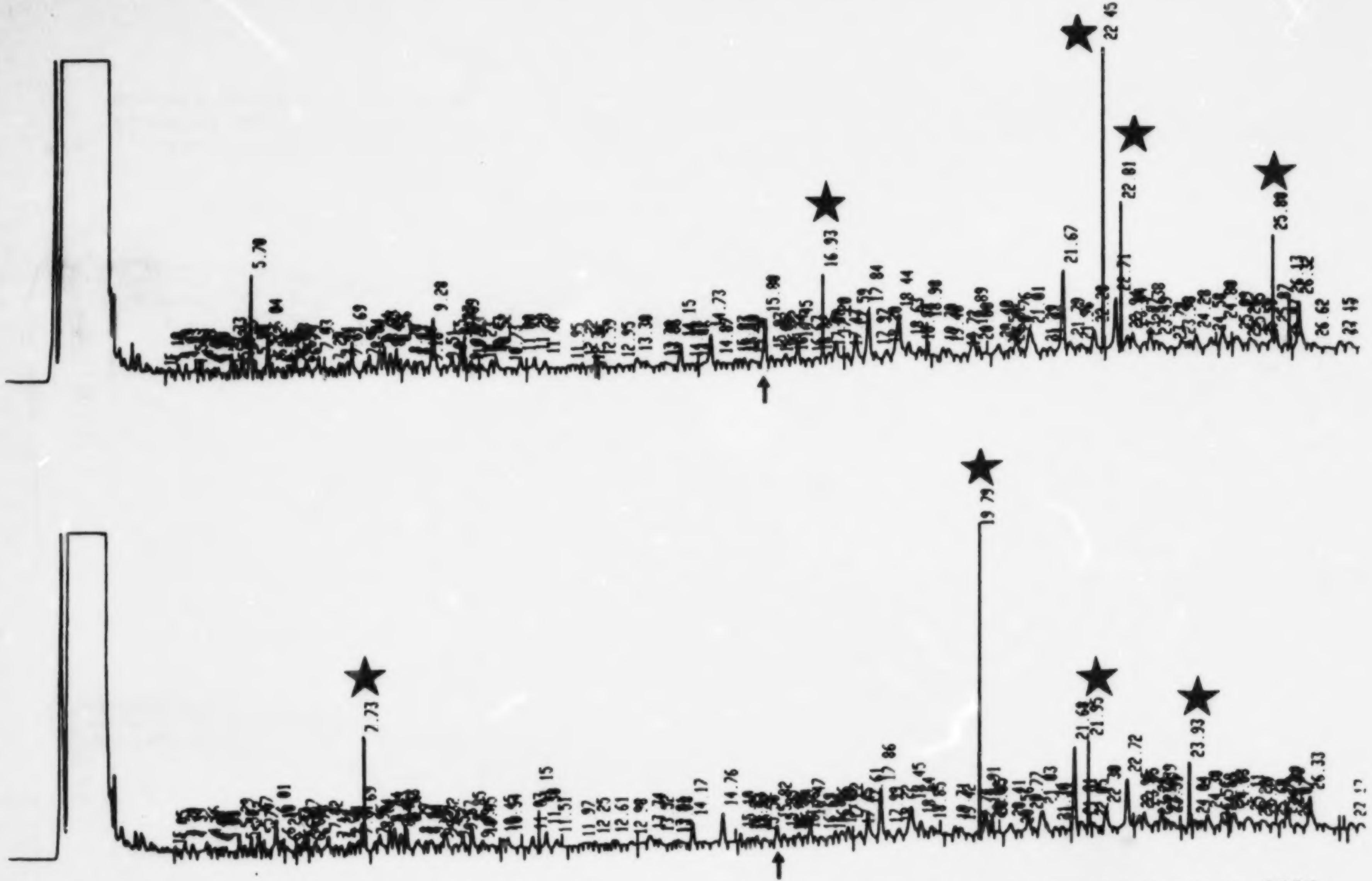


Fig. 2. Gas chromatograms of the hexane extract of Rossdale raw water collected March 4, 1986, with (top) and without (bottom) co-injection with geosmin standard on a 30 m DB-1 column. The geosmin peaks are noted with an arrow (↑), and noise spikes with a star (★).

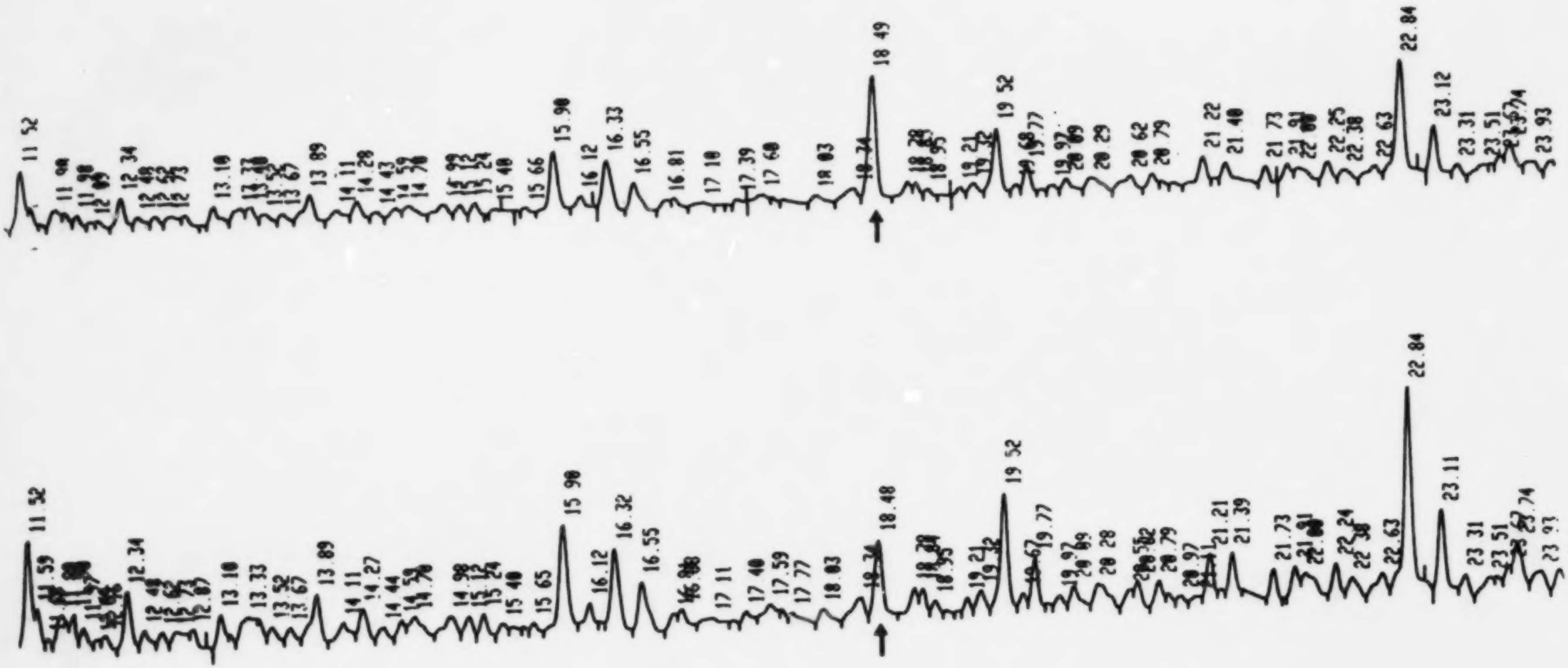


Fig. 3. Gas chromatograms of the hexane extract of Rossdale raw water collected March 4, 1986, with (top) and without (bottom) co-injection with geosmin standard on a 30 m DB-17 column. Geosmin peaks are noted with an arrow (↑).

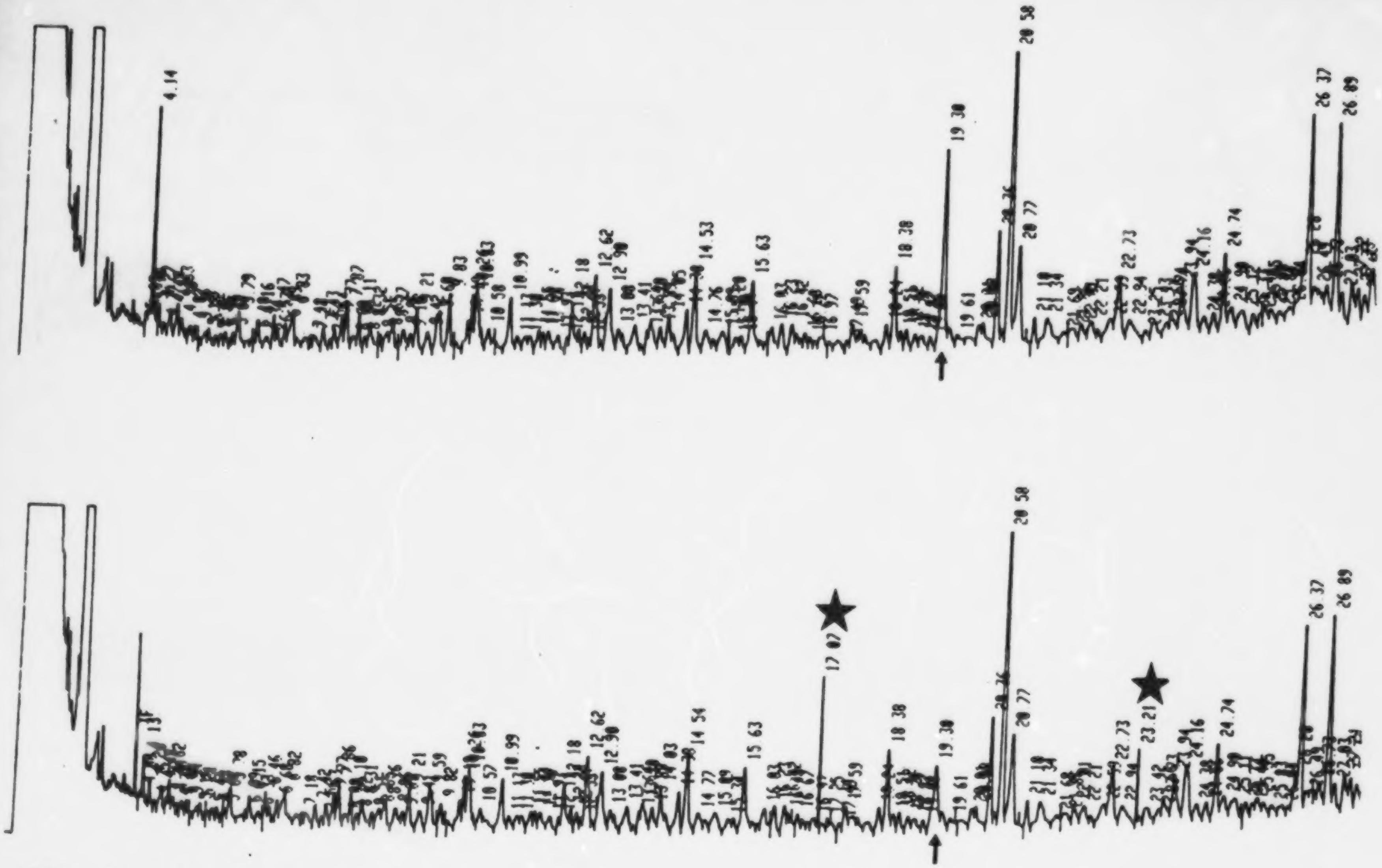
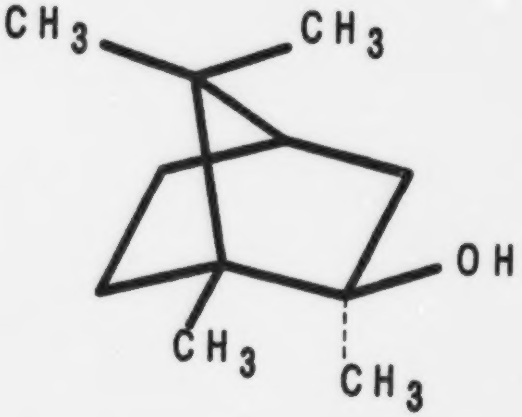


Fig. 4. Gas chromatograms of the hexane extract of Rossdale raw water collected March 4, 1986, with (top) and without (bottom) co-injection with geosmin standard on a 30 m Supelcowax 10 column. Geosmin peaks are noted with an arrow (↑), and noise spikes with a star (★).

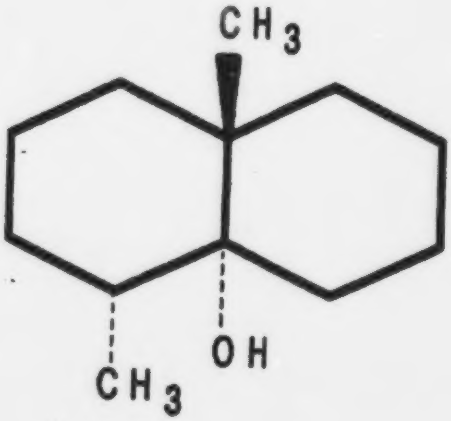
Appendix

The systematic names, and molecular and structural formulae for MIB and geosmin.

MIB is 1,2,7,7 tetramethyl-*exo*-bicyclo[2.2.1]heptan-2-ol, $C_{11}H_{20}O$, mol wt 168.



Geosmin is *trans*,*trans*-1,10-dimethyl-9-decalol, $C_{12}H_{22}O$, mol wt 182.



HEALTH EFFECTS THAT MIGHT BE ANTICIPATED FROM THE EDMONTON DRINKING WATER SUPPLY

A Report to

Steve E. Hrudey and Associates, Ltd.

Edmonton, Alberta
Canada

by

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June 29, 1986

INTRODUCTION

The City of Edmonton has experienced seasonal problems in delivering an aesthetically pleasing water to its consumers. The causes of the seasonal taste and odors that are encountered are complex and inextricably associated in a reciprocal way with the need to produce a water that protects the public from waterborne communicable disease. Consequently, it is essential that all aspects of the problem be critically assessed before remedial action is taken.

The present report deals with but one aspect of the overall problem, an assessment of the health effects that may be attributed to chemicals that have been found in Edmonton's drinking water in the past and identification of any problems that are foreseeable with some remedial measures that might be applied.

The issues identified and the recommendations that are made in this report should be interpreted in light of the information base available at the time. Since there is an obvious need to implement some corrective actions within the near future, it has not been possible for the author of this report to review all the relevant scientific literature that is available on each individual component that has been identified in Edmonton's drinking water. Neither was it practical to review all aspects of Edmonton's water system. For expedience, it was necessary to rely on a number of engineering assessments and reviews of the relevant literature that were prepared by others to arrive at the conclusions made. The following sources form the basis for this report:

1. Reports of mutagenicity test results by Carcinogen Testing Laboratory (3) and GeneBioChem, Ltd. (1) in various municipal

water supplies and under a variety of condtions.

2. Routine analyses from the Alberta Environment Treated Water Surveys (1983 to 1986) for organic and inorganic contaminants of the finished drinking water in Edmonton at different times of the year and at different locations within the system.
3. Hazard Assessment Documents prepared by the Canadian Centre for Toxicology that compare the dose-response characteristics of hazards that have been identified with individual chemicals with the concentrations that have occurred in Edmonton's drinking water.
4. A report entitled "Evaluation of Monitoring and Treatment for Protozoan Pathogens" authored by G. S. Logsdon of USEPA, Cincinnati, OH specifically for the current study.
5. A report by James M. Montgomery Engineers, Inc. on an analysis of the taste and odor problems in Edmonton by A. R. Trussell specifically for the current study.
6. A report "Pilot Scale Investigations of Alternative Drinking Water Treatment Processes at Edmonton, Alberta" by P. M. Huck et al., 1986.
7. An report by D. Gates documenting the ability of the water plant operators to measure residual levels of chlorine dioxide and chlorite in finished drinking water with associated data on measured residuals at the plants and in reservoirs.
8. A report "The Edmonton Drinking Water Study: Report and Recomendations Regarding Various Health Effects Questions" by Toxcon Consulting, Ltd done specifically for the current study.
9. "Formaldehyde and Other Aldehydes" National Academy Press, 1981

10. SNARLS produced by the Office of Drinking Water, USEPA.

11. A meeting held with representatives of the utility, the provincial government, a representative of the company marketing the ClO_2 generating system in Edmonton on May 23, 1986.

12. A summary of epidemiological statistics from National Health and Welfare Canada.

13. A draft manuscript entitled "Contaminants in drinking water and cancer risks in Canadian cities" by D.T. Wigle et al.

Other factual sources of information that were available in the author's literature files will be identified in the text and listed in the reference section of the report.

It should be understood that there are a great number of unknowns involved in judging the extent to which drinking waters might present subtle toxicological hazards to the consuming population. Most of these problems are by no means unique to Edmonton. First, we are limited by our analytical capabilities; only a small portion of the organic material in drinking waters derived from surface sources can be chemically identified. Much of this material is simply natural organic material with varying and complex structure. This natural organic material forms a precursor pool that reacts with added disinfectant to form by-products that are of some toxicological concern. A second problem is that an appropriate toxicological data base does not exist for many of the chemicals that have been identified in drinking water. This is particularly true of reaction products of disinfectants because some of these compounds are not produced commercially. These are very difficult and important problems that require further research. However, this lack of information should not be viewed with undue alarm. The information that is available (e.g. epidemiological data) would suggest that any additional hazard that might be associated with these variables is small, perhaps non-existent.

For purposes of clarity the report will be divided into the following sections:

1. Implications and interpretation of data that indicates the presence of mutagenic chemicals in Canadian drinking waters.
2. Comments on epidemiological investigations that have been conducted.
3. Review of compounds that have been identified in Edmonton drinking water and review the opinions of the Canadian Centre of Toxicology as to whether they pose unacceptable health hazards at the concentrations encountered.
4. Examine the processes involved in drinking water treatment to determine if they directly or indirectly introduce any unacceptable health risks that might be unusual if not specific to Edmonton.
5. Examine recommendations that have been made for research in the health area to determine which should be addressed by Edmonton and which problems represent a longer term national concern.
6. Summary and Conclusions.

I. Implications and Interpretation of Mutagenicity Test Results

The series of reports dealing with mutagenic activity in drinking waters follows the same pattern that has been experienced in a number Western countries since the mid-1970s. Essentially this pattern reflects the beginnings of an awareness of the true nature of drinking water contamination based on scientific information rather than speculation. Essentially this line of research began with the development of the Salmonella testor strains (i.e. the Ames' test) and related short-term tests provided a relatively simple means to detect mutagenic activity of chemicals. Shortly after their development these methods were applied to mixtures of chemicals that had been isolated from drinking waters. The initial finding of mutagenic activity in the Salmonella testor strains (e.g. Carcinogen Testing Laboratory Report No. 82-0411) was confirmed in tests conducted in eukaryotic organisms (such as yeast as in the GeneBioChem, Ltd. report). This was, in turn, followed by a recognition that the sampling techniques might be suspect (e.g. Carcinogen Testing Laboratory Report No. 84-0196). Finally, it was realized that most of the mutagenic activity observed in drinking waters comes about as a result of the disinfection process (particularly, but not exclusively chlorination) and is usually not the result of a contaminated source water (Carcinogen Testing Laboratory No 84-0196). Unfortunately, these reports are inconclusive as to whether the mutagenicity found in chlorinated waters arose from the treated water or were the result of mutagen formation by reaction of chlorine with the XAD-2 resin or some of its degradation products. On the other hand, there is plenty of confirmatory evidence in the general literature that chlorination produces mutagenic (e.g. Meier et al., 1983; Meier et al., 1985). For reasons outlined below, it is difficult to ascribe much meaning to these results in terms of human health effects. Consequently, I propose not to review these data in detail, but will concentrate on why it is best to interpret these results conservatively (in the scientific sense).

There are severe limits on how results from mutagenicity testing done only in *in vitro* systems can be applied to estimating human health hazards. These test systems have evolved to assume a couple very important functions in test protocols for carcinogenic and mutagenic chemicals. These are to 1.) identify chemicals for further study (i.e. those chemicals that possess a biological property that is important in chemically-induced cancer) and 2.) to determine if a chemical that has produced cancer in man or experimental animals has the genotoxic properties thought necessary for tumor initiation. Neither of these functions carries the requirement that the potency of the chemical in the *in vitro* system parallel its potency in experimental animals or man. In fact there is little evidence that such a relationship exists. Development of data that parallels the dose-response characteristics in man is the function of the higher level testing that is done *in vivo* in experimental animals. *In vivo* methods also have their shortcomings, but they do incorportate many aspects of human metabolism and function which are absent in the *in vitro* systems. Attempting to base judgements of risk on data strictly derived from *in vitro* tests frustrates the whole purpose of ordered testing rationales that are currently used in the regulation of toxic chemicals.

To properly fulfill the screening role, it is important that these *in vitro* test systems produce a minimum of false negative results so that there would be little chance of missing an important carcinogen. Given this requirement, it is not surprizing that these tests have a reasonably high false positive rate even when dealing with the testing of single chemicals. One must be even more concerned about this latter possibility when testing complex mixtures of concentrated organic material from surface water supplies.

A second issue that must be addressed in this type of study is that the sample interferes with the performance of the test. This can be manisfested by cytotoxicity to the test organism, a variable that has been taken into account in the studies reviewed here. A second possibility is that the activity of the metabolic activation system may be inhibited by the sample. There is evidence that humic acids do interfere in this manner (Meier and Bull, 1985), a fact that is rarely taken into account in studies of complex mixtures and was not in the present case. Consequently, the conclusion that indirect acting mutagens were not present in the water samples must be considered suspect.

Nevertheless, Edmonton residents can take some comfort in the fact that there is little evidence that mutagenic chemicals are present in their finished drinking water. This is primarily attributable to the use of chlorine dioxide and/or chloramine as disinfectants rather than chlorine. By way of contrast, the levels of mutagenic by-products observed in Bassano water are more typical of chlorinated supplies. In all liklihood, very high levels of mutagenic by-products and trihalomethanes would be produced in Edmonton water if chlorine were to be used as the disinfectant during the spring runoff period.

II. Comments on the Epidemiological Studies which have been Conducted.

From a strict scientific point of view, the most appropriate data for estimating hazards to human health is derived from human exposures. This is simply because it is data directly derived from the species of concern. Two types of data are theoretically possible; that derived from human experimentation and that obtained by studying populations of humans that have been exposed to the disease-causing agent in an occupational setting or involving other activities. The former situation is occasionally possible when one can be certain that the effect being studied is not life-threatening at the doses which will be employed and carries with it virtually no chance of irreversible damage. Unfortunately, these are not the kinds of chemicals that most often are of concern in drinking waters. Thus, we are forced to rely on epidemiological studies for most of the data that is directly taken from human experience. It is exceedingly important that these type of studies be continued not only for reasons of dealing directly with the problem at hand, but because they also form the basis for establishing the validity of test results in lower animals for estimating human health hazards. For a variety of reasons, this latter data is and will continue to be the major information base for estimating human health hazards that are associated with chemical exposures.

Unfortunately, there are definite limits to application of epidemiological results to assessment of risks. Because the human exposure is not controlled, proof of causality is very difficult. In acute disease epidemiology involving infectious agents one often has the opportunity to support epidemiological associations with direct measurements of the exposure and directly associate this with the disease involved in an outbreak. With chemically-induced chronic disease it is often very difficult to disassociate the effects of a particular chemical from other variables that are in the environment. The diseases produced are rarely unique to one chemical. As a result, a single epidemiological study is rarely viewed as convincing evidence that a chemical produces cancer (IARC, 1985) or other chronic disease.

Negative epidemiological studies present a different kind of problem (IARC, 1985). They must be considered in terms of their sensitivity which is always a function of the sample size and the dose that is encountered in the environment. In animals studies the lack of sensitivity can be compensated for by increasing the dose. In epidemiological studies the dose is not controlled by the investigator. Consequently, even the best designed epidemiological study that produces negative results can only be used to state that the risk involved is less than the upper limit of the sensitivity of the study in terms of the size of doses and populations studied. It is conceivable that a negative study could not exclude the possibility of a relative risk greater than unity (IARC, 1985). It is rare that epidemiology studies actually state the limits on their relative sensitivity in terms of the dose that was studied.

There appears to be very little epidemiological data that is available and relevant to the quality of Edmonton's drinking water. The data which is available do not identify any patterns of disease that that are peculiar to Edmonton that might be reasonably attributed to chemicals known to contaminate drinking water with the exception of cirrhosis of the liver. The concentrations of chemicals that could cause this disease are present in drinking water at ineffective doses. Alcohol consumption would be a more likely explanation for the higher incidence of this disease. In other cases, the age specific mortality ratios suggest that Edmonton is lower than most Canadian communities for most cancers. There is no information as to whether these differences are in any way related to drinking water variables. The higher incidence of death due to respiratory disease and motor vehicle accidents would not seem attributable to the water supply either.

The more general relationships between water variables in Canada (not specific to Edmonton) that have been identified in the Wigle et al., paper are similar to relationships found in the U. S. and elsewhere. A small increase in the risks of certain cancers of the gastrointestinal and genitourinary tracts seems to be associated with chlorination. However, it must be pointed out that ecological studies of this type do not adjust for a wide variety of confounding factors, so although some results are statistically significant not much weight can be assigned to such small age specific mortality ratios.

None of the above data are directly relatable to Edmonton's drinking water quality. Nevertheless, some comfort can be taken from the fact that there are no unusual patterns of chronic disease mortality that could be reasonably be associated with drinking water quality. Although these findings are controversial, the fact that chlorination seems associated with a higher incidence of certain types of cancers argues that Edmonton may be escaping some small degree of hazard from this quarter. However, it must be stated that most of these studies do not distinquish between chlorination and chloramination. Some studies along this line were contemplated in the Boston area, but to date I am aware of no results that have been published. In a draft report on a 90 day study in mice and rats (NTP,1982) increased mitotic figures, cellular hypertrophy and unusual chromatin patterns were seen in mice subjected to treatments of chloramine of 100 mg/L or more in their drinking water. It remains to be established whether these findings have any carcinogenic implications in the chronic phases of this study. I would point out that there have been no studies even conducted to determine whether the use of chlorine dioxide is associated with increased cancer risk.

In the final analysis, however, one must take the conservative view that there is no epidemiological evidence that Edmonton's water supply has contributed adversely to the public health.

III. Review of Hazards that Could be Anticipated from Individual Components Identified in Edmonton's Drinking Water.

The occurence of inorganic chemicals (disinfectant-related contaminants excluded) in the Edmonton drinking water exceeded established standards for these chemicals in drinking water only in one case. The exception was a maximum value reported for mercury seen at the Smith plant in 1984. Although samples taken at the Rossdale plant were below the standard, they were also elevated suggesting that this was a general phenomena, not one that was confined to the Smith plant. The form of mercury seen was not specified. It is assumed that the mercury standard of 1 ug/L was established because of concerns over methyl mercury rather than inorganic mercurials. Since the mean values for mercury did not exceed the standard in 1984, it is very unlikely that any adverse health effects resulted from this low level of mercury contamination. It is notable that mercury levels in both treatment plants have been consistently below detection limits since 1984. The information provided did not give an explanation for the higher mercury levels observed in 1984 nor any reasons why these levels have decreased to below detectable limits since that time. I presume the decreases were the result of some remedial action. If that is the case, there should be no concern in Edmonton about health effects that might arise from inorganic contaminants. If not, some investigation into possible sporadic sources of mercury into the water supply is warranted to alleviate concerns over occasional spikes of contamination that might occur without being detected at the usual sampling frequencies.

To my knowledge, there are no official standards that have been established for aluminum in drinking water. However, I note that the levels of aluminum seen in Edmonton's finished drinking water occasionally exceeds 1 mg/L. These levels appear to be high compared to other data I have seen. I note also that Logsdon pointed out some potential difficulties in the way alum is used in Edmonton. While there has been no causal link established, concern has been expressed over a potential relationship between aluminum in drinking water and senile dementia. Experiments in animals indicated that aluminum applied to the surface of the brain could produce some of the same pathological lesions. I know of no experiments that have confirmed the possibility of such effects via oral exposure. There does, however, appear some relationship between aluminum in water used for dialysis of patients in chronic renal failure and a similar disease referred to as dialysis dementia. I do not believe that protection of dialysis patients against aluminum in drinking water is necessarily the responsibility of the water utility, but that of the attending physician. In view of the general concern in this area (whether real or not) plus the questions Logsdon raised about the effectiveness of alum under the conditions employed in Edmonton, alterations in treatment practice that would result in lower aluminum levels would be prudent.

In general, organic chemicals that were identified in routine analysis of Edmonton's drinking water were present at such low levels that they would be of little concern on an individual basis. In a sense, total reliance on a chemical-by-chemical approach to define toxicological hazards in drinking water avoids the basic question of whether the water is safe or not because such a small fraction of the organic material present in drinking water sources can be identified and measured (estimated to be less than 1%). Again this is not a problem that is peculiar to Edmonton and one that cannot be simply resolved. However, there are some patterns to the contamination that should be taken into consideration. It is not necessary to have all this information to come to some remedies that avoid potential problems by the exercise of common sense.

There appear to be three different patterns associated with the occurence of organic contaminants of Edmonton's drinking water. There are compounds which tend to be present at both water plants at reasonably consistent concentrations [e.g. chloroform, bis(2-ethylhexyl)phthalate]. A second group of chemicals seems to be seasonally detected at both water plants (e.g. benzoic acid, benzeneacetaldehyde). Finally, there is a group of chemicals that occurs seasonally only at the Rossdale W.T.P. (e.g. xylenes). This latter situation indicates that there is something different about the inputs to the two water plants during part of the year even though the Saskatchewan river is the source water in both cases.

Judging from information presented at the meeting in Edmonton and that provided subsequent to the meeting, the number of sewer outfalls which are upstream from the Rossdale W.T.P. appears to be the critical issue. It is not unreasonable to assume that these outfalls are the source for xylenes. Recognizing that the methods are not available to characterize even a substantial fraction of the organic contaminants in drinking water, one must ask the question of what other chemicals might come from this source that would be undesireable contaminants of drinking water. While there is no reason to conclude that the levels of xylene observed in the water would produce any measureable health effects on the consuming population (USEPA SNARL = 2200 ug/L), the presence of the xylene clearly indicates a source of drinking water contamination whose impact on health probably cannot be addressed. The choice appears to further document and study the nature of that contamination and its health effects or to eliminate the source. I cannot comment upon the best course of action in this respect as I have no knowledge about the nature of other contaminants that might be present in the sewer outfalls. This is obviously a question that must be answered by people that are knowledgeable about these sewer outfalls. Common sense would suggest that removal of factors which make the system vulnerable might be the most cost-effective approach in the long term.

With respect to the chemicals detected upon routine analysis that impact both treatment plants, there is little to fear from a health effects standpoint. The trihalomethane levels are lower than would be expected in water supplies that use chlorine as the sole disinfectant. The relatively low levels present are largely attributable to the use of chlorine dioxide as the primary disinfectant. While chloroform concentrations may represent some appreciable risk (relative to other putative carcinogens that are present) the risk in Edmonton would have to be considered less than the average for North American cities. Using the data obtained from the mouse in the NCI bioassay for chloroform (controversial because this result was not repeated when chloroform was administered at comparable doses in drinking water) the upper 95% confidence limit of the risk at concentrations encountered in Edmonton would be approximately one additional case in a lifetime in a population of 100,000 people (calculated using the Multistage Model). The other chemicals detected occurred at very low concentrations and only sporadically. For example, benzene was observed at a concentration of 5 ug/L in one sample (December, 1984), but most often was below the detection limit of 0.1 ug/L. Benzene is a recognized human carcinogen, but the upper 95% confidence limit of risk at the level of detection would only be about three additional cases of leukemia per lifetime per 10,000,000 population. Dichloromethane (methylene chloride) was also detected in one sample at a level of 23 ug/L, but generally was found not detectable (limit of detection 10 ug/L). Using worst case assumptions (upper 95% confidence interval of the estimate, plus that methylene chloride is present at the detection limit at all at other times in the year), an increased risk of about 5 additional cancer cases in a lifetime in a population of 1,000,000 people is calculated. Similar calculations for trichloroethylene and tetrachloroethylene (which were observed in only one sample at 0.4 ug/L and 3 samples at 0.2 ug/L, respectively) would indicate a risk of of about 4 additional cases per lifetime per 10,000,000 population and 3 cases in 100,000,000, respectively. Bis(2-ethylhexyl)phthalate was detected at concentrations of up to 7.8 ug/L with an average concentration of 2.5 ug/L at the Rossdale plant which would represent a carcinogenic risk of about 4 additional cases per 100,000,000 population. The other chemicals identified (toluene, ethylbenzene, xylene) have not been identified as carcinogens and their concentrations are orders of magnitude below those which have been proposed as guidance numbers.

The above represents essentially the worst case analysis of the hazards that might be associated with the chemicals that have been encountered in Edmonton's drinking water. The model used to calculate these risks (i.e. the multistage model) makes a series of assumptions about the mechanisms involved in chemical carcinogenesis. The most important of these assumptions being that carcinogens act through a irreversible step considered many scientists to involve a somatic mutation. There is evidence to suggest that most of the compounds identified in Edmonton's water that have produced cancer in experimental animals have very little genotoxic activity. The use of this assumption probably results in higher estimates or risk for these compounds. Adding this to the use of the upper 95% confidence interval of the estimated risk almost guarantees that the levels of risk are much lower than stated above. Non-carcinogenic chemicals identified in Edmonton's drinking water are present at such low concentrations that no adverse health effects could reasonably anticipated. While there were trace quantities of chemicals that are putative carcinogens, the risk levels that can be projected using a worst case analysis are vanishingly small. To put this into context, the estimate of risk that would be derived by adding up all the risks indicated above would be approximately an order of magnitude less than the risk permitted by the WHO guideline for chloroform in drinking water. In general, one would have to conclude that the levels of risk that would be anticipated from the organic chemicals identified in Edmonton's water is considerably less than would be calculated for most cities that use chlorine for the purposes of disinfecting drinking water.

The biggest question that remains is how to deal with the potential health effects of the organic chemicals in drinking water are not detected in routine chemical analyses. Even if there are no overt health effects that can be projected, it is clear that during a brief period each year Edmonton's water is something less than palatable. A more detailed analysis of water using non-conventional techniques obtained March 5, 1986 (Brander Gardens sample, analyzed by Coutts, University of Alberta) emphasizes this point. During this period the dissolved organic carbon increases from normal values of 1-2 mg/L to more than 20 mg/L. Consequently, the analyses may not be applicable at other times of the year. Therefore, they have not been specifically considered in this report with respect to any potential health effects. Compounds that were detected by this method included a variety of organic acids, aldehydes, ketones, esters, ethers, steroids, amides and hydrocarbons not otherwise detected. The point is that with expensive, non-standard techniques a much wider variety of organic chemicals were detected at estimated concentrations of 1 to 30 ug/L. While these concentrations are still low in an absolute sense, they are relatively high compared to the components discussed above.

It is useful to note that some of the compounds, particularly the aldehydes, that have been identified in specialize analyses do have properties which make them respiratory irritants (Steinhagen and Barrow, 1984). The sensory irritation could account for some of the complaints registered during the spring runoff period.

Although there was inadequate data available to assess the health effects of some of the components identified in these latter analyses, there is some reason to be concerned. For example, n-hexane is recognized as a potent neurotoxin that appears to act through a metabolite, 2,5-hexanedione. This activity is very specific to the spacing between the two ketone groups. While neither of these two compounds were identified in the sample, compounds with similar structure or which might be metabolized to a similar toxic intermediate were identified. Similarly, simple aldehydes such as formaldehyde and acetaldehyde have been identified as chemical carcinogens in animals (IARC, 1985). There is no data of which I am aware that would indicate that the aldehydes actually identified in Edmonton water are carcinogenic. Therefore, there should be no undue concern within the context of available health effects information and the relatively low concentrations that were observed. If other considerations (e.g. taste and odor) dictate remedial action for this particular group of constituents, some additional, but presently non-quantifiable benefit could be realized through the removal of related compounds that are potentially harmful.

With respect to the non-conventional organics analyses, some effort should be expended to determine which if any of these compounds are responsible for the taste and odor problems that are encountered during the spring runoff. It is extremely important that the chemicals responsible for these problems and their source within the system be established so that remedial action can be taken. Even if there are no adverse health effects that could be anticipated from these compounds, public confidence in the utility will not be established until this problem is solved.

By way of a summary for this section, I believe that the Edmonton water works should look very seriously at the pattern of contamination with respect to the seasons and to the sources that would contribute chemicals to the two treatment plants. Based upon these considerations one can draw up a variety of scenarios about what else could come from these sources that might not be obvious from routine chemical analyses. If there are chemicals that will enter either treatment plant that are not desireable from considerations of health effects or aesthetics then cost-effective remedial action should be taken. I am not an engineer, a cost analyst or a politician so I am not qualified to make recommendations as to the most appropriate remedy. From a health effects point of view, however, the location of the Rossdale water intake has to be considered less than desireable. I am very uncomfortable with what is an essentially uncontrolled input into this system from the storm sewer outfalls. Unless that plant employs very sophisticated treatment processes that can act as efficient barriers to essentially any microbiological or chemical challenge that could arise from these outfalls Edmonton's drinking water will remain vulnerable.

IV. Potential Contributions of Drinking Water Treatment to Adverse Health Effects.

The issue that sparked the concerns over the quality of Edmonton's drinking water are seasonal taste and odor problems. These appear to be of two basic sources; a brief intense problem associated with spring runoff that is observed at both water treatment plants and problem that also occurs in the spring, but is observed only at the Rossdale W.T.P.. The sewer outfalls upstream of the Rossdale plant seem strongly implicated in the latter problem. This problem has essentially been dealt with in the previous section. The former situation has some serious implications for the treatment processes that are utilized to produce a microbiologically safe water.

This year the spring runoff was associated with an order of magnitude increase in dissolved organic carbon (DOC) in the raw water and a 2 to 3 fold increase in the DOC in the finished water at both plants. Such heavy organic loads produce unusual demands upon the disinfectant. This is especially true in Edmonton where there is some concern over a microbiological contaminant that is resistant to disinfectants, *Giardia*. Effective killing of *Giardia* cysts requires substantial contact times to some minimum concentration of disinfectant (Logsdon). To meet the demand and obtain a contact time that is sufficiently long to inactivate the cysts much higher concentrations of disinfectant are necessary. This in turn leads to the formation of by-products that may of potential concern. The formation of trihalomethanes when chlorine is used as the disinfectant has received the most attention. The formation of chlorite as a by-product when chlorine dioxide is used as the disinfectant presents a much more acute problem than trihalomethane formation with chlorine.

In Edmonton, there also seems to be fairly convincing circumstantial evidence that chlorine dioxide increases taste and odor problems (irritating and "hydrocarbon" odors are described) when the dose is increased. As will probably be reported by others this experience with chlorine dioxide is not limited to Edmonton. I recently learned that Newport News, VA has suspended the use of chlorine dioxide because of a number of consumers complain of irritation to the eyes and nose when chlorine dioxide doses have been increased above 1 mg/L (Robert Hoehn, personal communication). Somewhat similar problems evidently have been encountered using ozone as the disinfectant in other water supplies.

Because of the high organic load, substitution of chlorine for chlorine dioxide during this period of high organic load would undoubtedly produce unacceptable levels of trihalomethanes during this period plus introducing a different type of taste and odor problem. Disinfection with chloramine would decrease the formation of trihalomethanes. However, as is pointed out by Logsdon, chloramine is not likely to provide adequate protection against _Giardia_ outbreaks with the lack of a predistribution storage capacity in Edmonton.

In the remainder of this section, it is my intention to briefly discuss some of the health problems that could be anticipated from _inappropriate_ use chlorine dioxide as the primary disinfectant. It is assumed that this agent will continue to be used in Edmonton. Although specific levels of disinfectants and by-products may be mentioned, these are not meant to supercede any guidance numbers that have been issued by the Provincial government. Research efforts are continuing to identify the hazards that might be associated with chlorine dioxide and its by-products and it is likely that official guidance in this area will change in the future. Therefore, it would be wise to consult the appropriate authorities before any substantial investments are made in changing current practice.

Because of its reactive nature, it is difficult to determine whether toxicological effects of chlorine dioxide are attributable the parent compound or a reaction product. Similar problems exist with other disinfectants. The best defined and simplest reaction product of chlorine dioxide is chlorite. Organic by-products are undoubtedly produced _in vivo_ as well as in drinking water, but to date, none of these compounds have been identified as being responsible for any of the toxicological effects of chlorine dioxide. As will be discussed below, some of the effects of chlorine dioxide have been attributed to the formation of chlorite when it is ingested. This conclusion has most often been formed on the basis that chlorite is more potent in producing certain effects than is chlorine dioxide and the fact that chlorite and chloride are the two major inorganic by-products that can be observed in the urine of animals administered chlorine dioxide whereas none of the parent compound is recovered (Abdel-Rahman et al., 1980 and 1982). This type of evidence is not conclusive, but this logic will be used in discussing the toxicological effects of chlorine dioxide and its by-products below.

In at least one case, depressed thyroid function (Bercz et al., 1982) it is apparent that chlorine dioxide produces an effect at lower doses than any of its inorganic by-products. This does not exclude the possibility that an organic reaction product produced in the gastrointestinal tract is responsible for this effect. However, the conclusion is the same, there is reason to indict chlorine dioxide as being toxicologically important that can be separated from its conversion to chlorite. This effect of chlorine dioxide was first observed in the African green monkey (Bercz et al., 1982), but has since been documented in rat pups (Orme et al., 1985) and found to be associated with delays in brain development (Taylor and Pfohl, 1985). This effect was observed at a dose of 9.5 mg/kg/day in the primate, but was absent when administered at a dose of 3.5 mg/kg/day for 4 weeks. In the rat, the effect was observed at a dose of 14 mg/kg/day, and behavioral effects associated with the depressed thyroid function disappeared at about 3 mg/kg/day.

Haag (1949) in studies financed by Olin, found that chlorine dioxide administered in drinking water at 100 mg/L (12.5 to 13.4 mg/kg/day) decreased survival of albino rats in a chronic study. The survival of the animals was not affected at concentrations providing a dose of 1.3 mg/kg/day. In a repeated dose study in humans, Lubbers et al., found no effect associated with drinking 500 ml of water containing 5 mg chlorine dioxide per liter for a period of 12 weeks, providing a dose of 0.036 mg/kg/day. If one applies the usual safety factors of 100 to the most sensitive of the animal studies (decreased longevity) man's dose should not exceed 0.013 mg/kg per day. If the usual factor of 10 is applied to the negative work done in humans a limit of 0.0036 mg/kg/day is derived. Using the USEPA's assumption that a 10 kg child consuming 1 liter/day is the most critical factor (an assumption supported by the fact that thyroid function was depressed in rat pups when chlorine dioxide was via the lactating dam without affecting her thyroid function) the residual level of chlorine dioxide should not exceed 0.13 mg/L. The recommendation of shorter term limits by NAS (1982) considered only the human data (Lubbers et al., 1981) which was negative for all disinfectants and did not consider the substantially greater exposures that were necessary to actually produce adverse effects in animals. In other words, this work provides an underestimate of the actual NOAEL of unknown dimensions. It would be inappropriate in my view to restrict the use of any of the disinfectants based solely on a study that failed to demonstrate any effects at all. Rather these results in humans should be used to support the values arrived at from data in experimental animals where actual effects were demonstrated.

The principal toxic effect that has been observed with chlorite to date is the production of hemolytic anemia. Although it also produces methemoglobinemia, this effect requires higher doses administered in a bolus. Evidence of decreased glutathione levels could be seen in rats administered doses of chlorite as low as 5 mg/kg/day and increased red cell turnover was observed in the cat at doses as low as 3 mg/kg/day (Heffernan et al, 1979). These effects were not observed at doses of 1 mg/kg/day and 0.6 mg/kg/day respectively. Studies which reported sporadic decreases in these parameters at lower doses have been disregarded since the authors employed inappropriate statistical tests. In the human study (Lubbers et al., 1981) a dose of 0.036 mg/kg/day administered over a 12 week period was without effect. Haag (1949) also observed some renal changes in rats (a complication frequently observed with hemolytic agents) at a dose of 9.3 mg/kg/day which was not observed at a dose of 0.7 mg/kg/day. Choosing the NOAEL established in the cat of 0.6 mg/kg/day and applying the usual safety factor of 100 brings one to the conclusion that humans should not be exposed to more than 0.006 mg chlorite/kg body weight. Applying this to the 10 kg child consuming 1 liter of water (justified by the sensitivity of this age group to oxidants), the concentration of chlorite in drinking water should not exceed 0.06 mg/L. If the rat NOAEL (probably more appropriate) is used, the corresponding level would be 0.1 mg/L.

The above exercise is consistent with USEPA policy, but does not necessarily mean that these are the levels that will be chosen as standards for chlorine dioxide or chlorite. Although some rationale is articulated for the safety factors of 10 and 100 used above, the selection of these values is somewhat arbitrary. Consequently, the Agency could depart from these levels if they feel it can be justified.

I ran through this exercise for two reasons. The first is to document that there is an independent component to the toxicities of chlorine dioxide and chlorite suggesting that independent standards should be established (i.e. the thyroid effect appears independent of the hemolytic effect). Of course the hemolytic effects of the two agents are probably not independent, but by the time one calculates the conversion of chlorine dioxide to chlorite vs. other species, the incremental risk would be quite small. The second reason is to impress upon the people in Edmonton that there are geniune limits to the amount of chlorine dioxide that should be added to drinking water. The actual number established as the limit depends upon the chance one is willing to take that an acute illness will result. I emphasize the word acute because the effects we are worried about with chlorite are acute hazards that will be induced by short term excursions over a critical dose. It is not a case where average doses over a lifetime is important as is presumed the case for chemical carcinogens. I will also point out that there is no question that humans are susceptible to agents that produce hemolytic anemia so the question of whether the effects extrapolate across species or not is not an issue. To put this in perspective, the same cannot be said for trihalomethane-induced cancer. In the case of the anti-thyroid effects of chlorine dioxide the effects of concern would have to be exerted over some time period to result in irreversible effects. In the developing human, the critical period of exposure (during pregnancy or in the postnatal period) could be relatively short (weeks or months) whereas in the adult thyroid function would have to be depressed for some time (months to years) before an irreversible effect would become manisfest (e.g. thyroid cancer would be a possibility).

For the above reasons, I was very happy to see that Edmonton waterworks is now placing considerable emphasis on the measurement of chlorine dioxide and chlorite residuals in the water provided to the consumer. The movement of the point of chlorine dioxide application downstream would also appear likely to decrease the dose of disinfectant required. However, there appears to be some conflict between the amounts of disinfectant that are needed to protect the system against contamination by Giardia and the need to minimize the dose of disinfectant from the standpoint of producing toxicological problems.

The recent experience in Edmonton (which I am beginning to get a feeling is not confined to Edmonton) with taste and odors generated by the use of chlorine dioxide should provide some impetus into looking at the by-product issue with chlorine dioxide in the same way that it has been examined with chlorine. While there is little specific reason for suspecting that these by-products have produced serious health effects, this arises more from the lack of data rather than any serious evaluation of the problem. Now that it seems clear that there are some difficulties associated with the by-products of chlorine dioxide, it is hoped that research will be stimulated to characterize the toxicological properties of some of the by-products that are formed as well as their contribution to taste and odor problems.

The above discussion and my previous comments (letter to S. E. Hrudey dated 3/22/86) indicate a high degree of concern about situations where the water treatment plant operators are tempted to always meet disinfectant demand with chlorine dioxide. After the discussions that were held in Edmonton, it should be clear to everyone that it is important to limit the concentrations of both chlorine dioxide and chlorite that are present at the first consumer's tap. Above, a case has been made that these two compounds should not be regulated on a total oxidant or combined basis because the toxicology of the two agents are different (i.e. 2 mg of chlorite does not equal 1 mg of chlorine dioxide and 1 mg of chlorite either qualitatively or quantitatively in toxicological terms). The point at which the limits should be set is a matter I think best left to the responsible authorities in Canada.

The questions identified above need to be extended to other chemicals that are used in drinking water treatment. As indicated in the Trussell report, it is important to be aware of the quality of bulk chemicals that are used in water treatment and to be aware of some of the problems that arise from residues of products that gain access to the finished water. This will be addressed further in the section containing the recommendations of this report. However, water plant operators and managers should be aware that the National Sanitation Foundation, the American Water Works Association Research Foundation and State agencies are beginning a certification program aimed at dealing with such problems with products that are introduced into or contact potable water. This program replaces the activity of USEPA that was suspended 6-7 years ago. EPA never had legislative authority in this area, so all the so-called approvals were simply advice to state and local governments. The only official regulation in this are that will be put into place in this area in the U.S. are MCL's for contaminants of finished water that are introduced as the result of using these chemicals. This effort is just now getting underway and I don't anticipate much in the way of formal regulations for a number of years.

V. Recommendations for Research in the Health Effects Area.

There are a variety of research questions that should be pursued with respect to the quality of Edmonton's water supply. For the sake of simplicity, I will separate these into two categories; those which are peculiar to Edmonton and are primarily of local concern and those which are problems that are generic to producing drinking water from surface sources and are probably more efficiently done at a provincial or national level. Some of the former questions will be somewhat broader than recommendations for health research as such but will point out that chemical and biological characterizations of the problem are fundamental to making assessments of potential health hazards.

A. Research questions that are of peculiar concern to Edmonton.

1. Identification of the chemicals by-products that are produced through reaction of chlorine dioxide with the high organic load that arises during the Spring runoff from the upland bogs which feed the Saskatchewan River. Most critically in the short term are those compounds which are responsible for the taste and odors that result. However, it is also important to be certain that some of the residual by-products that may remain after the taste and odor problem is addressed do not have any adverse health effects. Perhaps the phase of this work that would be most appropriately done by Edmonton is the identification of these by-products. If insufficient health effects information exists for these chemicals, it is very important that those chemicals which are produced at

appreciable concentrations in the finished drinking water be characterized toxicologically. A minimum level of characterization would be subchronic studies in two species, reproductive and teratology studies and a battery of in vitro tests. The function of the latter tests is to determine whether lifetime studies for chemical carcinogenic activity is warranted. Whether this latter work should be financed by the city or some higher level of government is a matter that I cannot make specific recommendations on. In the U.S. that question would depend primarily upon how specific the problem is to a certain water supply. If the effort could be shown to address a national rather than a regional or local concern then the federal government was justified in addressing the problem. The question of how unique the use of chlorine dioxide is to Edmonton would play a key role in this area. In the U. S., the Toxic Substances Control Act (TSCA) could be brought to bear on such an issue and the manufacturer would be held responsible for demonstrating the safety of established chemicals under Section IV of the Act. Alternatively, pesticide legislation might be invoked.

2. An epidemiological study should be conducted in Edmonton if chlorine dioxide continues to be used as the primary disinfectant. Despite the fact that a few cities may be using chlorine dioxide, it use has never been subjected to the epidemiological scrutiny that chlorine has. As mentioned earlier in this report, the use of chloramine is also undergoing some scrutiny because of its widespread use in the U.S. The only two investigations that have been conducted in studying health outcomes from the use of chlorine

dioxide were grossly inadequate. If Edmonton could conduct such a study, it should concentrate on sensitive populations such as young children and peoples of Mediterranean extraction who have enzyme deficiencies that render them more sensitive to hemolytic agents. Again, one must ask the question of whether this is Edmonton's problem or a national problem. As mentioned above, it is my belief that the company manufacturing and marketing products for this type of use could be held responsible for such studies under section IV of the Toxic Substances Control Act (TSCA). I do not know if a comparable authority exists under Canadian law.

3. The adequacy of the treatment train, including the disinfectant, to deal with Giardia needs to be demonstrated. The discussions held in Edmonton raised serious questions in my mind as to whether there was sufficient contact time at adequate levels of disinfectant to inactivate Giardia cysts during the spring episodes. My reading of Logsdon's report raises questions about the adequacy of protection against Giardia with other operations in the plant. It is regrettable that a Water Utility as large as the one serving Edmonton does not pilot plant facilities nor adequate personnel to address problems as they arise in the source water. Given that circumstance the whole operation is a rather large and expensive experiment whose final product is the water that is being consumed that day! About all that can be done is to make your best guess and listen for the phone to ring. I think this has to be frustrating to a professional staff that is obviously trying to do a good job.

4. Some consideration should be given to developing a sampling scheme in Edmonton's distribution system that is more comprehensive than at present. This sampling could be done at a relatively low frequency (e.g. annually) and be confined to parameters likely to change in the distribution system because of differnet materials in mains and piping or differences in residence time within the system. Currently, sampling is done at only one tap served by each treatment plant and this is sufficient to characterize the extent to which contaminants of the source water reach the consumer. However, a number of problems in drinking water arise in its distribution. Some of the heavy metals found in drinking water arise almost exclusively from pipe (e.g. Pb). Similar problems exist with leaching of asbestos from A/C pipe. In a similar vein, disinfection reactions continue in the distribution system. Cross-connections and regrowth of microorganisms in the system provide new substrates to produce more and perhaps different reaction products. Chlorine dioxide-induced taste and odor problems encountered in Newport News, VA were greatly intensified by overnight standing, suggesting that much of their problem arises in the distribution system rather than in the treatment plant (Robert Hoehn, personal communication). Consequently, meeting a standard at the treatment plant is not particularly meaningful in these cases.

5. In the Toxcon Consulting, Ltd. report, a recommendation was made concerning problems that might be associated with corrosivity of Edmonton's drinking water. Judging from the data I have seen, Edmonton's water does not appear to be corrosive. Therefore, I do

not see any reason to be concerned about this issue beyond the general point of knowing what is going on the distribution system made above. However, it could be of concern if the City is considering softening the water at the treatment plants.

B. Research Problems that Should be of Interest at Higher Levels of Government.

The report prepared by Toxicon Consulting, Ltd. suggested that substantial efforts be placed on determining whether fluoride is carcinogenic or not. This is an example of a health effects problem which has implications that apply much more generally than to Edmonton alone. It happens that I do not share the views nor do I endorse the approach suggested by the author of this report for fluoride. Nevertheless, supplementations of fluoride provided in drinking water continue to be an issue. However, the controversy is not particularly well grounded in science. As a result, I doubt that the issue will be decided on scientific grounds. Nevertheless, if there are doubts that about the safety of a particular process involved in water treatment that can be resolved by appropriate experimentation, it should be done. The basic conclusion of the epidemiological data that bears on fluoride exposure and cancer is negative and I personally do not believe pursuing the issue at this level of investigation will be profitable.

As to the *in vitro* evidence that is quoted to support the need for addtional studies on fluoride carcinogenesis, I have the following comments. The mechanism by which sodium fluoride produced morphological and neoplastic transformation in cultured Syrian hamster embryo cells and human oral keratinocytes is not clear. The means by which a "genotoxic" effect is or could be involved with this compound is not clear. I would suggest that the morphological changes observed might be more closely related to fluoride's well known effects on cyclic nucleotide metabolism. There is also evidence to suggest that chromosomal breakage occurs in cells as a normal step in differentiation, a process that may well be influenced by cyclic nucleotide levels within the cell. But given the author's point that the tests are positive, the approach should be to assess the carcinogenic effects of the chemical in lifetime studies, not to proceed with further _in vitro_ testing. This type of investigation is likely to be beyond the resources and capabilities of the City of Edmonton and, as I point out above, is the type of problem that should be pursued more generally.

I see some reasons to elaborate on or take exception to some of the other conclusions made in the report by Toxcon Consulting, Ltd. which I detail below. These points are not solely based on toxicological grounds; they include some consideration of practical limits to the responsibilities of a waterworks to the community. The items are identified below by the numbers provided in the text of that report (p. 20-21):

5. I do not think any argument can be made that the concentration of Na in Edmonton's drinking water represents a risk factor to any sensitive populations. An average of 4.2 mg/L is well below any guidance numbers provided for this very purpose. I do not think that it is reasonable to expect the water treatment plant or the City of Edmonton can take responsibility for individuals who choose

to use water softeners. This should be the responsibility of the individual and perhaps his physician.

6. I saw no indication that the pesticides indicated in this comment compounds were actually in Edmonton's drinking water. Consequently, speculation about their interactive toxicities is irrelevant to the problem at hand.

7. The levels of Pb and Se are well below any official standards that are currently on the books. You should be aware that there has been and will continue to be pressure to lower the standard for Pb in drinking water in the U. S.. The factor most responsible for this is that low doses of Pb are apparently absorbed much more efficiently than higher doses. Coupled with the relatively low "safety factor" that exists between current Pb exposures and levels that can be shown to produce subtle effects in man and experimental animals. I would not be surprised to see the drinking water standard for Pb move gradually towards 10 ug/L or lower in the future.

9. Again, phenols in source waters are not peculiar to Edmonton. Historically, these compounds have been looked for in a source water primarily because of taste and odor concerns they produce in the finished water. Since the determination of phenol was by the non-specific colorimetric method, it is not possible to deal with this result from a toxicological point of view. Judging from the nature of the contamination you are experiencing, I suspect phenol

contamination is mostly of natural origin, although the possibility of an industrial source should be explored. The Toxcon report is, however, correct when he says that the toxicology of phenols has been poorly characterized. Again, unless there is a specific industrial source, this problem is a broad one in water supply that is not peculiar to Edmonton.

10. I do not believe that the studies quoted support the notion that chlorine dioxide or chlorite were teratogenic. These studies included inadequate numbers of animals to address this issue. In selected cases, follow-up studies have been conducted by other investigators (i.e. B. D. Carleton, Battelle, Columbus, OH has done some studies which are yet to be published).

11. I do not understand the author's conclusion that the risk to the developing fetus due to cyanide is low but obviously not zero. I did not see any information quoted which would indicate that there was any risk associated with cyanide levels that have been found in Edmonton's drinking water.

14. I do not understand why the beneficial effects of fluoride must be specifically demonstrated in Alberta. These effects have been well documented in many areas.

16. My views on the fluoride issue were identified at the beginning of this section.

17. The write-up on fluoride is not sufficiently detailed to identify the doses of fluoride that are involved in producing osteosclerosis and thyroid malfunction. It is implied that doses of fluoride will cause accumulation of fluoride in bone, a desireable effect to a point. Excessive fluoride may be associated with these effects. If these diseases are not produced in other areas that use 1 mg/L fluoride supplementation, I doubt that there is any case to study this relationship in Edmonton. In any case, this is a broader question than should be addressed more broadly than in Edmonton if it has any merit.

18. The only drinking water constituent that has been identified with ALS is Pb. At the low levels of Pb encountered in Edmonton, I do not think this is an area to worry about.

Recommendations of the Toxcon Consulting, Ltd. report (p. 27-28) are reviewed in the same fashion:

1 & 2. I endorse the recommendation for mounting a monitoring program that is directed at measuring constituents at the tap as mentioned earlier in this report. The sampling scheme should be developed to consider differing materials in the distribution system, including household plumbing, and varying residence time in the distribution system. These analyses should be limited to parameters that might be expected to change in the distribution system (don't forget microbiological parameters). The frequency of such sampling should be related to periods of time when variations

in the quality of source or treated water is changing (e.g. monitoring for disinfectant by-products during the spring runoff and a suitable control period) as well as a fixed schedule of relatively low frequency (e.g. once annually). If changes in water quality is found not to vary these parameters significantly, the scheduled monitoring might be suspended unless major changes in treatment are anticipated (e.g. softening).

3, 4, 5, & 6. These are rather extraneous subjects for research. I find it hard to believe that these are peculiar problems to Edmonton. Research of this kind has been sponsored and reported upon in the literature.

8 & 9. I have detailed my thoughts on fluoride above.

11. I have serious reservations about the validity of results that could be obtained from the 8 individuals identified in the Toxcon report. It constitutes an admittedly biased sample. I cannot conceive of a control population that would not be equally biased (i.e. complainers against non-complainers). If this work is undertaken, it should be for other than scientific reasons (e.g. public relations).

VI. Summary and Conclusions

It is obvious that Edmonton has periodic problems with the quality of its drinking water. As unpleasant as that problem is to the consumer, there is little objective reason to suspect that the chemical contamination associated with these episodes has produced any adverse effects on health. This conclusion is, of course, qualified by the limited amount of information that is available that bears directly on the issue. On the other hand, it should be pointed out that the information that has been assembled for evaluation of Edmonton's water approaches that which is possible within the limited period of time available. It is important that work begin to address some of the obvious ills in the system based upon the information that is available. At the same time, I hope that Edmonton accepts the responsibility for better characterizing their source water and their treatment of it so that as problems arise in the future there is an objective data base that can be used to solve these new problems.

Although it is not possible to point to individual chemicals at the concentrations encountered in Edmonton drinking water as being responsible for adverse health effects, there are several situations which exist in the Edmonton system that could easily allow chemicals and/or microbial agents into the system at concentrations that are sufficiently high to produce adverse health effects. Of these, inadequate control of the disinfectant application is undoubtedly the most critical and in my view the most likely to produce adverse health effects if not strictly controlled. The remaining areas also carry some risks, but they are more reflective of good water treatment practice that protect the water from unanticipated problems rather than being addressed at a particular health effects issue.

Based upon these concerns, I have developed the following set of recommendations:

1. It is extremely important that dosage of chlorine dioxide and the contact time be carefully controlled and monitored. On the one hand, it is essential to maintain some minimum residual level for a minimum contact time to protect against waterborne infectious disease. On the other, there is a limit to what can be added because of the toxicological hazards associated with chlorine dioxide, chlorite and in all probability some of the organic by-products that are produced. This becomes an acute problem during the spring runoff period when substantial increases in the load of natural organic chemicals in the river occur, increasing in turn the amount of disinfectant that must be applied to achieve the minimum contact time. Alternate forms of disinfection do not appear as attractive alternatives in this situation. Logsdon's report recommends approaches that could perhaps help to remove this problem. However, if the competing needs to protect against toxic levels of disinfectant and/or by-products and to provide microbiologically safe water cannot be resolved, treatment to remove the organic material from the source water may be the sole alternative. In this recommendation, I have avoided making a specific recommendation on the maximum levels of chlorine dioxide, chlorite and other by-products that should be allowed because I see this as a governmental responsibility that should involve input from more than one source. However, I have provided some indication of how such numbers would ordinarily be developed in other regulatory

arenas that may be used as general guidance.

2. Although no specific health effects could be anticipated from the contamination of water that was experienced at the Rossdale plant, there should be little comfort taken from this fact. This is an obviously vulnerable point in Edmonton's water system that may allow the introduction of chemical and biological contamination that could escape routine monitoring. It should be recognized that there is probably an unrecognized input into the system from storm sewers throughout the year. Uncontrolled inputs into this kind of system has been known to cause severe problems in sewage treatment plants (e.g. in Louisville sewage treatment plant a few years ago, a number of workers were overcome by fumes of hexachlorocyclopentadiene in the plant because a trucker decided to clean out a tank truck and the wash water entered a storm sewer). If a large scale contamination enters the source water upstream from a drinking water intake, the effect may not be confined to waste treatment plant operators.

3. It is very important from both the health effects and public relations points of view to do as much as is possible to identify the by-products which are formed with disinfection of Edmonton's drinking water during normal conditions on the Saskatchewan River as well as during the periods of high organic load from upland runoff during the Spring. Obtaining the comparative data for these different time periods will indicate whether the problem is simply the result of quantitative variations of the source water quality

during different times of the year or if there are qualitative differences in the products formed as well. In my view, this would have important implications in any long term remedial actions that the City would take, particularly if any health effects are associated with these by-products that would not be automatically taken care of by control of the taste and odor problems. Even if the issue ends up focussing only on taste and odor problems, these data would be useful in deciding whether seasonal measures would suffice to control the problem or whether the whole treatment process needs to be altered because the problem is lurking just below the threshold at other times of the year. In brief, these data should be used to:

a. Review the literature for potential health effects of any by-products that are identified and set appropriate standards.

b. Conduct preliminary toxicological studies for those compounds which lack an adequate data base (should include _in vivo_ subchronic studies and some preliminary _in vitro_ mutagenicity screening at a minimum). If these studies indicate the possibility of a problem, then longer studies and broader based (e.g. reproductive and teratogenesis studies) studies may be necessary. I don't think I can comment on the question of the party who should be responsible for these studies because that is a question of Canadian law and convention.

c. Provide the data base that will be required to evaluate corrective actions that will obviously have to be taken. I'm afraid that if this problem is addressed purely on empirical grounds and solved in one year, the city will run the risk of a repeated episode down the line. If a recurrence happens after the solution has been reported to be found, it will be very difficult to restore public confidence in the system.

4. A quality assurance and information base should be developed for chemicals that are used in the treatment of drinking water. I noted in the report by Trussell, that is apparently no effort to confirm the quality of bulk chemicals used in the treatment plants. It is extremely important, in my view, that the managers of water treatment operations become sensitized concerning the nature of the materials that are used in treatment and their quality. There have been a number of products used in water treatment in the past that certainly would not measure up to today's standards for saftey (e.g. lead pipe and coatings, coal tar paints, A/C pipe). New problems have been identified with modern products (e.g. the carcinogenicity of acrylamide, epichlorohydrin and other monomeric components used in coagulant aids, contamination of chlorine gas with carbon tetrachloride, etc.) and questions of understanding and controlling certain current practices in drinking water treatment (e.g. alum, disinfection, etc.). The use of these products and processes all have some health effects implications. It is as important to recognize what remains in the finished water as it is to know what is added (e.g. the use of any coagulant aid for drinking water

treatment also introduces lower molecular weight material that will end up in the finished water despite the fact that the polymer might be quantitatively removed during coagulation, settling or filtration. Therefore, the content of low molecular weight material in these products is very important). Consequently, it is essential that those managing the water treatment plant be as up to date on these issues as is possible. This does not mean that they have to be practicing toxicologists, but simply that they are prepared for the fact that changes in the information base in this area can have as great an impact on water treatment as the introduction of new treatment processes. This recommendation fits in with Logsdon's recommendations concerning operator training and the development of professional contacts. These kinds of efforts are extremely important to expectations that water plant operators are capable of dealing efficiently with problems that are inevitable in a system as large as Edmonton's. The city cannot and should not depend solely upon vendors of equipment or other products for this kind of information.

5. The monitoring system for Edmonton's drinking water quality should include more than one sampling point in the distribution system, preferrably at the taps in selected homes. This sampling could include consideration of homes from which complaints arise, but the sampling scheme should not be predicated on this question. Rather, it should concentrate on variables that are inherent in the Edmonton distribution system (e.g. areas of high and low flow, houses with different plumbing materials, mains with different

materials or water contact surfaces, long distribution lines, etc.). The parameters chosen for this monitoring should be limited to those chemicals (and microbial agents) that might be expected to increase in the distribution system. There should be no reason to look at source contaminants (e.g. pesticides). Except for special circumstances (e.g. by-product formation during spring run-off), these analysis should be conducted at a relatively low frequency (e.g. annually). Once the system has been characterized, this type of monitoring might be reduced and/or eliminated. Routine monitoring at a single tap for each treatment plant should be sufficient to establish whether contaminants in the source are reaching the consumer in dangerous amounts or not.

6. Consider other means of preventing contamination of the finished water supply by Giardia cysts so that the system is not entirely dependent upon chlorine dioxide disinfection. This might be an alternative to organics removal as a means of reducing disinfectant demand during the spring runoff (item 1). In no case, however, should the water system be rendered vulnerable to waterborne infectious disease. Personally, I would go to chlorine disinfection and accept the trihalomethane risk for a short period of time before I would accept risks associated with a vulnerable system or overdosing with chlorine dioxide.

7. As mentioned in an earlier section, I believe that an epidemiological study should be conducted that focusses on the use of chlorine dioxide as a primary drinking water disinfectant. There

is essentially no substantive data that bears on this issue in the literature. The effects that one would expect and the sensitive populations to the kinds of effects that would be predicted would probably be well represented in a community as large as Edmonton. If possible, Edmonton should be paired against at least one city with similar demographics that utilizes chlorine for drinking water disinfection and another which uses either chloramine or ozone. Again, the question is whether this should be done by Edmonton or some higher governmental authority is a question that must be decided in Canada.

References:

Bercz, J. P., Hones, L., Garner, L., Murray, D., Ludwig, D. A. and Boston, J. (1982) Subchronic toxicity of chlorine dioxide and related compounds in drinking water in the nonhuman primate. Environ. Health Persp. 46:47-55.

Haag, H. B. (1949) The effect on rats of chronic administration of sodium chlorite and chlorine dioxide in the drinking water. Report to the Mathiseson Alkali Works from H. B. Haag of the Medical College of Virginia. Dated Feb. 7, 1949.

Heffernan, W. P., Guion, C. and Bull, R. J. (1979a) Oxidative damage to the erythrocyte induced by sodium chlorite, *in vivo*. J. Environ. Pathol. Toxicol. 2:1487-1499.

Heffernan, W. P., Guion, C. and Bull, R. J. (1979b) Oxidative damage to the erythrocyte induced by sodium chlorite, in vitro. J. Environ. Pathol. Toxicol. 2:1501-1510.

IARC (1985) IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. International Agency for Research on Cancer, Lyon, France. General principles applied by the working group in evaluating carcinogenic risk of chemicals or complex mixtures, pp. 13-34; Aldehydes pp. 101-177.

Lubbers, J. R., Chauhan, S. and Bianchine, J. R. (1981) Controlled clinical evaluations of chlorine dioxide, chlorite and chlorate in man. Fundam. Appl. Toxicol. 1:334-338.

Meier, J. R., Lingg, R. D. and Bull, R. J. (1983) Formation of mutagens following chlorination of humic acid: a model for mutagen formation during drinking water treatment. Mutation Res. 118:25-41.

Meier, J. R., Ringhand, H. P., Coleman, W. E., Munch, J. W., Streicher, R. P., Kaylor, W. H. and Schenck, K. M. (1985) Identification of mutagenic compounds formed during chlorination of humic acid. Mutation Res. 157:111-122.

Meier, J. R. and Bull, R. J. (1985) Mutagenic properties of drinking water disinfectants and by-products. In: Jolley et al., eds. Water Chlorination: Chemistry, Environmental Impact and Health Effects. Lewis Publishers, Inc. Ann Arbor, MI., pp.207-220.

NTP (1982) Subchronic study of monochloramine in the Fisher 344 rat and the B6C3F1 mouse. Draft report of the National Toxicology Program, National Institute for Environmental Health Sciences, Research Triangle Park, NC.

Orme, J., Taylor, D. H., Laurie, R. D. and Bull, R. J. (1985) Effects of chlorine dioxide on thyroid function in neonatal rats. J. Toxicol. Environ. Health 15:315-322.

Steinhagen, W. H. and Barrow, C. S. (1984) Sensory irritation structure-activity study of inhaled aldehydes in B6C3F1 and Swiss-Webster mice. Toxicol. Appl. Pharmacol. 72:495-503.

Taylor, D. H. and Pfohl, R. J. (1985) Effects of chlorine dioxide on neurobehavioral development of rats. In: Jolley et al., eds. Water Chlorination: Chemistry, Environmental Impact and Health Effects. Lewis Publishers, Inc. Ann Arbor, MI., pp. 355-364.

HEALTH ASSESSMENT OF SELECTED CHEMICALS FOUND IN DRINKING WATER IN THE CITY OF EDMONTON.

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HEALTH ASSESSMENT OF SELECTED CHEMICALS FOUND IN DRINKING WATER IN THE CITY OF EDMONTON.

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INTRODUCTION

The number of substances which have been or might be expected to be found in the drinking water of a city such as Edmonton, and thus could have been included in this report, is very large. The focus of this health assessment was therefore restricted by the judicious selection, on the basis of existing knowledge of toxicology and potential for human exposure, of a group of substances of priority concern. This list is not intended to be comprehensive but is a compromise between the availability of resources and concern for the substances involved.

In the case of some substances, toxicity profiles and assessments such as those published by the US National Academy of Sciences already existed but required updating. Other profiles were developed specifically for this project. The profiles developed here are organized in similar fashion to those of the National Academy of Sciences and are based on literature published up to the end of 1985, with more recent data included where it became known. In order to reduce the length of these profiles, they were focussed on drinking water or at least oral exposure where that was possible. In addition, only those studies judged to be pivotal have been discussed in detail; those of lesser importance were included for completeness but not discussed at length.

1. NAME: BENZOIC ACID

1.1. CAS No.: 65-85-0

1.2. SOURCES AND USES

The major use of benzoic acid is as a food additive. It is used as a preservative and antimicrobial agent in a wide variety of food products including soft drinks and tinned fruit. Benzoic acid is also used as an intermediate in the manufacture of benzoates and benzoyl compounds. Benzoic acid occurs in nature in free and combined forms. Most berries contain appreciable amounts (approximately 0.05%) (Windholz, 1983). Benzoic acid has been identified as an essential oil constituent of Paeoniae radix, where it has a faintly urine-like odour (Miyazawa, 1984). No data were found on levels of benzoic acid found in drinking water supplies.

1.3. PHARMACOLOGY AND METABOLISM

Benzoic acid is rapidly absorbed and exists in the blood in the free state, not bound to proteins. Benzoic acid does not undergo oxidation in the animal body but combines with glycine or glucuronic acid, forming, respectively, hippuric acid and benzoylglucuronic acid (WHO, 1983). The rat is closest to man as far as the metabolism of benzoate is concerned. In man, rabbit and rat, benzoic acid is almost entirely excreted as hippuric acid, whereas dogs excrete more conjugated glucuronic acid than hippuric acid (Williams, 1959). Studies with sodium benzoate-1-C^{14}, administered intraperitoneally, orally, or subcutaneously to the rat, showed that quantitative excretion occurs in the urine within one to two days, with less than 1% of radioactivity appearing in the feces (Lang and Lang, 1956). Accumulation does not occur. One healthy man given 6, 9, 13.9, 34.7, and 69.3 mmole of sodium benzoate showed a complete elimination within 10-14 hours (Schachter, 1957). Studies with male Wistar Albino rats, administered benzoic acid by gavage, established that 0.1% of the applied dose was retained in the tissue (Freitag, 1985). The maximum urinary excretory rate is dependant on the availability of glycine for conjugation. Benzoic acid is also an intermediate metabolite of phenylalanine, tyrosine and quinic acid (WHO, 1983). WHO (1983) stated that benzoic acid is effectively and rapidly metabolized and eliminated by the body without apparent tissue injury.

Benzoic acid is a specific powerful inhibitor of the D-amino-acid oxidase. As well, benzoic acid inhibits pepsin digestion and sodium benzoate inhibits trypsin digestion of fibrin, but they have no effect on amylase or lipase (WHO, 1983). Avigan et al. (1955) reported that concentrations in the range of 10^{-3} M resulted in some unspecific inhibitory effects on the metabolism of fatty acids. Benzoate is a hydroxyl radical scavenger and Weitberg et al. (1985) suggested that, in the future, benzoate supplementation of target cells may play a role in protection from oxygen radical-generated chromosomal injury.

1.4. ANIMAL TOXICOLOGY

1.4.1. ACUTE

The following acute toxicity data were found in the literature:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Sodium	rat	?	oral	LD_{50}	2700	Deuel et al, 1954 benzoate
	rat	?	IV	LD_{50}	1714	Spector, 1956
	rabbit	?	oral	LD_{50}	2000	Spector, 1956
	rabbit	?	SC	LD_{50}	2000	Spector, 1956
	dog	?	oral	LD_{50}	2000	Spector, 1956
Benzoic acid	rat	?	oral	LD_{50}	2000-2500	Ignat'ev, 1965

A case of benzoic acid poisoning affecting 28 cats has been documented (Bedford et al, 1971). The cats ingested meat containing 2.39% benzoic acid. The effects were nervousness, excitability, convulsions and loss of balance and vision. Autopsies showed damage to intestinal mucosa and liver. The authors (Bedford et al, 1971) suggested that the sensitivity of the cat may be due to its failure to form benzoyl glucuronide.

1.4.2. CHRONIC

In a recent study Sodemoto et al. (1980) fed 10 male and 10 female rats sodium benzoate in the diet at levels of 0, 0.5, 1, 2, 4, or 8% for a period of six weeks. All rats on the 8% level and 19 rats on the 4% level died within four weeks. A significant reduction of body weight gain was noted only in the 8 and 4% groups. Animals treated with sodium benzoate showed hypersensitivity as an acute toxic effect, but convulsions or other symptoms were not seen. Survivors showed no morphological abnormalities at sacrifice.

After reviewing the literature, WHO (1983) concluded that long-term toxicity studies have demonstrated exposure to benzoic acid in the diet at a level of 1% (500 mg/kg) did not cause observable toxic effects (WHO, 1983).

1.4.3. MUTAGENICITY AND GENOTOXICITY

Ishidate *et al*. (1983) examined the mutagenicity of benzoic acid in the Ames *Salmonella* assay using *S. typhimurium* strains TA92, TA1535, TA100, TA1537, TA94 and TA98. Benzoic acid was negative for mutagenicity when incubated with the S-9 fraction at a maximum dose of 10 mg/plate. Sodemoto and Enomoto (1980) also reported that benzoic acid was negative in the Ames *Salmonella* assay. The sodium salt of benzoic acid was also tested in the Ames *Salmonella* assay. Results were negative for the highest non-cytotoxic dose (3 mg/plate). Ishidate *et al*. (1983) also tested benzoic acid and sodium benzoate for their ability to induce chromosomal aberrations in Chinese hamster fibroblasts (48-h exposure). The incidence of polyploid cells as well as of cells with structural chromosomal aberrations were recorded. The results for benzoic acid were equivocal, with a 1% incidence of polyploid cells and an 8% incidence of cells with structural chromosomal aberrations. Sodium benzoate was positive for chromosomal aberrations with a 1% incidence of polyploid cells and a 38% incidence of cells with structural chromosomal aberrations.

Njaga and Gopalan (1982) observed that sodium benzoate at concentrations ranging from 0.05×10^2 to 5×10^4 ppm induced cytological effects on *Vicia faba* root mitotic cells, including inhibition of DNA synthesis and the induction of anaphase bridges and subsequent micronuclei.

1.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Minor and Becker (1971) injected rats intraperitoneally with sodium benzoate at dose levels of 100, 315, or 1000 mg/kg during days 9-11 or 12-14 of gestation. An increase in *in utero* deaths and reduction of fetal body weight was seen for both treatment periods at the 1000 mg/kg dose level. Undefined gross malformations were observed in the group exposed to 1000 mg/kg on days 9-11 of gestation. This study suffered several shortcomings, including exceptionally high dose levels and a failure to define the types and frequency of gross malformations. As a result, meaningful conclusions can not be drawn from this study.

Sodium benzoate produced no teratogenic effects in chicken embryos after injection of 5 mg/egg into the air cell of eggs on day 4 of incubation (Verrett _et_ _al_, 1980).

1.4.5. CARCINOGENICITY

In a 24-month study, male and female Fischer-344 rats were fed diets containing 1 or 2% sodium benzoate. No evidence of carcinogenicity was demonstrated (Sodemoto and Enomoto, 1980). Marquardt (1960) also reported negative carcinogenicity results for rats fed diets containing 0.5 and 1% benzoic acid in the diet over their entire lifespan. Studies with other laboratory animals were not found.

1.5. HUMAN TOXICOLOGY

Tolerance to benzoic acid appears to vary in man. Some can tolerate 24-40 g while others suffer marked gastrointestinal; disturbances after ingestion of 5.7 g sodium benzoate (WHO. 1983). For many years, a liver function test was used in man based on the urinary excretion of hippuric acid after a test dose of benzoic acid (6 g orally, or 1.5-2.0 g intravenously). The "lethal dose low", defined as the lowest dose of a substance introduced by any route reported to have caused death in humans, is reported as 500 mg benzoic acid/kg body weight (oral) (Sax, 1984).

Acute toxicity in man is readily reversible and probably due to a disturbance in acid-base equilibrium rather than associated with any tissue damage (WHO, 1983).

1.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

Benzoic acid has been used as a food additive for many years and the established acceptable daily intake for man is 0-5 mg benzoic acid/kg body weight. Benzoic acid is defined as the sum of benzoic acid plus its sodium, potassium and calcium salts (WHO, 1983).

1.6. HAZARD ASSESSMENT

Benzoic acid was generally reported from Edmonton drinking water at levels less than 1 ppb, however, in March of 1986 levels of 9, 14, 16 and 49 ug/L were reported. The level of 49 ug/L corresponds to a worst case exposure of 1.66 ug/kg/day (60 kg man, 2 L/day). This value is some 3000 fold less than the ADI of 0-5 mg/kg. In addition, as benzoic acid is used as a direct food additive, and occurs naturally in some fruits, intake from food is probably a more significant source of benzoic acid than water. Based on the above considerations it appears that levels of benzoic acid present in Edmonton drinking water do not present a human health hazard.

2. NAME: BENZENE

2.1. CAS No.: 71-42-2

2.2. SOURCES AND USES

Benzene is produced as a byproduct of the petroleum industry and is used in the chemical industry as an intermediate in the production of a large number of chemicals such as styrene, cyclohexane, detergents and some pesticides. As well, benzene is used as a solvent. It is found in gasoline at levels usually <5% and has been reported in the drinking water of US cities at levels between 0.1 and 0.3 ug/L. The highest concentration reported in water was 10 ug/L from a US city (NAS, 1979).

2.3. PHARMACOLOGY AND METABOLISM

Benzene appears to be rapidly metabolized and excreted in animals and man (NAS, 1979). Products of benzene metabolism have been reported as catechol, phenol, hydroquinone, and hydroxyquinol which are eliminated in the urine as sulphate esters or glucuronides and phenyl-mercapturic acid. Benzene appears to be rapidly absorbed via inhalation or the oral route and is rapidly distributed throughout the body where it concentrates in organs and tissues in proportion to their lipid content (Greenlee et al., 1981 and Snyder et al., 1983). Bone marrow, an important target tissue has a high tissue partition coefficient for benzene. It has been suggested that the metabolism of benzene is important in the hematotoxicity, however little is known of metabolism in bone marrow (NAS, 1979).

2.4. ANIMAL TOXICOLOGY

2.4.1. ACUTE

Exposure to high concentrations of benzene vapors (20000 ppm) have lead to death in humans (EC, 1984). Selected toxicity data is given below:

Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Benzene	Rabbit	?	Inhl.	LC_{50}	3333 mg/m^3	EC, 1984
	Rat	?	Inhl.	LC_{50}	16000 ppm (4 h)	EC, 1984
					10000 ppm (7 h)	EC, 1984
	Mouse	?	Inhl.	LC_{50}	9980 ppm	EC, 1984
	Rat	?	Oral	LD_{50}	5600 to 3000	EC, 1984
	Mouse	?	Oral	LD_{50}	4700	EC, 1984

Studies reported by Kimura et al. (1971) showed that young rats were about 4 times more sensitive to benzene toxicity than older rats. Single exposures to high but non-lethal levels of benzene do not lead to the development of lasting harmful effects.

2.4.2. CHRONIC

The most commonly reported effect of chronic exposure to benzene in laboratory animals is that of leukopenia. Rozen and Snyder (1985) reported decreases in total lymphocytes, red cells and granulocytes in animals exposed to 300 ppm benzene after exposure periods ranging from 6 to 115 days. Inhalation exposure to 61 ppm benzene (5 h per day, 4 days per week) for 6 or more weeks resulted in a decrease in the number of leukocytes (Deichmann et al., 1963). However, it has been suggested that normal diurnal fluctuations in cell numbers in the blood may be misinterpreted as leukopenia and that reports of effects at concentrations below 1000 ppm may not stand up to critical statistical analysis (Laskin and Goldstein, 1977).

Wolf et al. (1956) reported that oral administration of benzene to rats at doses of 1, 10, 50 and 100 mg/kg body weight resulted in leukopenia and erythrocytopenia with a minimal effect level of 10 mg/kg body weight. Chronic exposure in humans has been reported to result in effects on the nervous system including reduction in peripheral nerve conduction velocity (Baslo and Aksoy, 1982). Inflammation of the respiratory mucosa, congestion of the kidneys and cerebral edema have been reported in cases of high exposure in humans. Prolonged occupational exposure to benzene at levels >100 ppm has been reported to produce aplastic anemia, leukopenia and thrombocytopenia and exposure to levels of ca. 25 ppm is reported to cause a reversible increase in red blood cell volume (IARC, 1982).

2.4.3. MUTAGENICITY AND GENOTOXICITY

A number of toxic effects on bone marrow cells have been reported in laboratory animals such as rats. The effects include changes in chromosome number and chromosome breakage, however, Laskin and Goldstein (1977) have suggested that these are not dose dependent and may therefore not be of major significance. In vivo cytogenetic effects of benzene have been well documented with rodents: increased micronuclei production, chromosomal aberrations and enhanced sister chromosome exchanges have been consistently observed (NTP, 1986). With rodents, males are generally more susceptible than females to the genotoxic effects of benzene. Ames Salmonella typhimurium mutation assays have not shown mutagenicity even in the presence of S9 fraction from uninduced and induced rat livers (Lyon, 1976). Other test systems have given mostly negative results but clastogenic effects have been reported in humans exposed to high occupational levels (IARC, 1982).

2.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Malformations of the palate and teeth were reported by Watanabe and Yoshida (1970) in the young of mice given subcutaneous injections of benzene at 3 ml/kg on days 11 to 15 of gestation. Delayed ossification of sternabrae and missing sternabrae have been observed in the fetuses of rats exposed to air containing 50 to 2200 ppm benzene (NTP, 1986). In contrast to this (IARC, 1982) it has been reported that exposure of pregnant rabbits to 500 ppm benzene resulted in no malformations. No direct teratogenic effects have been attributed to benzene and it is suggested that reports of malformations are related to maternal toxicity rather than direct effects on the fetus (IARC, 1982).

2.4.5. CARCINOGENICITY

Rozen and Snyder (1985) observed one case of hematocytoblastic leukemia and one case of plasmocytoma in C57BL/6 mice exposed to 300 ppm benzene for 480 days. CD-1 mice exposed to benzene at levels of 300 ppm benzene (6 h per day, 5 days per week) showed two cases of myologenous leukemia after 27 weeks of exposure (Goldstein et al, 1982). In another study on C57BL/6 mice, Cronkite et al. (1984) exposed animals to 300 ppm benzene (6 h per day, 5 days per week) for 16 weeks showed 6/90 animals with thymic lymphomas and 2/90 with unspecified lymphomas. Control animals did not show leukemia. Maltoni et al. (1983) dosed rats by gavage with 0, 50 and 250 mg/kg body weight benzene (5 times per week for 52 weeks) and reported dose related increases in zymbal cell carcinoma and mammary carcinoma in the females. Leukemia incidence was not increased. The National Toxicology Program carried out two-year carcinogenesis and toxicology studies on benzene with F344/N rats and B6C3F_1 mice with dose levels of 0, 50, 100, and 200 mg/kg body weight benzene administered by gavage. Male and female F344/N rats developed significantly increased incidences of primary neoplasms in the zymbal gland and oral cavity. As well, primary neoplasms were detected at above the control level on the skin of male F344/N rats. Male and female B6C3F_1 mice showed significantly increased incidences of neoplasms at the following sites:

zymbal gland, lymphoma, lung and harderian gland. Further, increased incidences of ovarian and mammary gland neoplasia were observed in the female $B6C3F_1$ mice while increased incidences of preputial gland neoplasia was observed in the male $B6C3F_1$ mice. NTP concluded that, based on their results, there was clear evidence for the carcinogenicity of benzene in male and female F344/N rats and for male and female $B6C3F_1$ mice.

Some benzene metabolites, namely phenol, catechol, hydroquinone, and p-quinone are considered carcinogenic (NTP, 1986).

2.5. HUMAN TOXICOLOGY

Several epidemiological studies on benzene have been conducted. However, in many of these studies, benzene was not the only solvent that the workers were exposed to. Vigliani (1976) conducted a study in which exposure to benzene at levels of 25 to 600 ppm in air was related to a 20-fold increased risk of leukemia. Infante et al. (1977) studied a group of workers exposed to levels of benzene below 100 ppm and suggested a 10-fold increased risk of myeloid and monocytic leukemia in the exposed group. Rinsky et al. (1986) reported an excess of leukemia in workers exposed to benzene at levels within the limits acceptable at the time of exposure (10 ppm). Unfortunately, none of these studies presented accurate exposure levels and extrapolation to lower doses is therefore impossible. Nevertheless, IARC (IARC, 1982) has stated that benzene causes damage to the hematopoietic system and increases the incidence of leukemia in humans. Single exposures to high but non-lethal levels of benzene can lead to loss of consciousness, irregular heartbeat, dizziness, headache, and nausea (NTP, 1986).

2.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

The USA-ACGIH TWA 8 hour day TLV exposure level is 10 ppm (EC, 1984) while the PEL and MAC are 10 and 100 ppm respectively (EC, 1984). The Occupational Health and Safety Administration proposed the adoption of 1 ppm as the "lowest feasible level" but this has not yet been accepted. (NAS, 1982). Suggested no adverse response levels (SNARLs) for drinking water calculated from the acute animal toxicity data were as follows (NAS, 1980 and 1982):

24 h exposure	12.6 mg/L	NAS, 1980
7 d exposure	0.25	NAS, 1982

In 1982, NAS (1982) withdrew the above 24 h SNARL. Chronic exposure SNARLs were not calculated because benzene is a putative leukemogen.

2.6. HAZARD ASSESSMENT

In its assessment of exposure to benzene in drinking water, NAS (NAS, 1982) considered the proportion of exposure form other sources. They estimated daily food intake of 250 ug/day and an intake from urban atmosphere of 600 ug/day. On this basis they calculated that exposure from water would contribute only 0.3% of the daily dose. Levels of benzene reported from Edmonton drinking water were normally less than the detection threshold of 0.1 ppb (0.1 ug/L) but was reported at levels above this on three occasions, the highest being 5 ug/L in March 1984. A worst case average exposure to 0.2 ug/day from water would, on the basis of the above, be equivalent to 0.025 % of total exposure from all sources and the human health risk in this context is probably negligible. Concern should be directed towards identification and control of the sources of contamination which result in the occasional reporting of levels above the detection threshold.

3. NAME: TOLUENE

3.1. CAS No.: 108-88-3

3.2. SOURCES AND USES

Toluene is used in the manufacture of solvents, a number of benzene derivatives, caprolactam, dyes and TNT. It is used as an additive to gasoline and is also formed during the process of petroleum refining and the distillation of coal tar. Toluene has been reported from water in a number of US locations at levels of up to 11ug/L (NAS, 1977).

3.3. PHARMACOLOGY AND METABOLISM

Toluene is absorbed most easily via the gastrointestinal and inhalation routes. No information on skin absorption was found in the literature except for the suggestion that it is similar to that for benzene (Wahlberg, 1976). Once taken up in the body, toluene tends to accumulate in body fat (Sato et al., 1974). This reservoir is of importance as it has been shown that saturation of fat-rich tissues such as brain does not occur even after 4 h exposure of mice to concentrations of 4000 ppm in air (Bruckner and Peterson, 1976). This factor has also been noted in man where subjects with least adipose tissue also had the smallest retention of inhaled dose (Carlsson and Lindqvist, 1977).

Up to 80% of toluene taken into the body is excreted in urine as hippuric acid and most of the remainder is exhaled. Less than 1% was excreted as o- or p-cresol and small amounts of benzyl alcohol were detected in hydrolysed urine (NAS, 1977). Measurement of hippuric acid has been used as an indicator of exposure but the quantitative value of this has been questioned because of individual variations in rates of hippuric acid excretion (Engstrom et al., 1976).

3.4. ANIMAL TOXICOLOGY

3.4.1. ACUTE

Most of the acute toxicity assays on toluene have made use of inhalation as a route of exposure. Selected values are given below.

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Toluene	Man	M	Inhl	Psychological effects	100ppm	Patty, 1981
	Monkey	?	Inhl	Psychotropic	100	ITII, 1981
	Rat	?	Inhl	LC_{50}	8000 4h	Patty, 1981
		?	Oral	LD_{50}	7000 mg/kg	" "
	Mouse	?	Inhl	LC_{50}	5300 8h	NRC, 1981
	Guinea	?	Oral	Non-toxic	1.2 ml/kg	Divincenzo and Krasavage, 1974

Patty (1981) reports an oral LD_{50} of 6.4 ml/kg in young adult rats and oral LD_{50} of 3 ml/kg in 14-day-old rats suggesting that young animals are more susceptible to its toxic action. Short term exposure of rats to toluene vapour (in the range of 1000-12000 ppm) has been reported to cause a slight decrease in liver microsomal enzyme function (Mungikar and Pawar, 1976), reduction of ability in tests based on animal performance (Bruckner and Peterson, 1978) and changes in sleep cycle (Takeuchi and Hisanaga, 1977).

3.4.2. CHRONIC

Rats exposed to toluene vapours at 1000 ppm, 8 h/d for 5 days (40000 ppm hours) showed increases in liver enzyme activity and acidosis (Tahti et al., 1977). Rats exposed to concentrations from 30 to 1000 ppm, 6 h/d, 5 d/week for 13 weeks (390000 ppm hours) showed no effects in clinical measurements of histological parameters (Rhudy et al., 1978). Fisher rats exposed to toluene at concentrations up to 300 ppm for 6 h/day 5 days/week for 6 months (234000 ppm hours) failed to show any symptoms of toxicity except for a lower rate of weight gain at the high concentrations (CIIT, 1978) Mice exposed to concentrations up to 1000 ppm for 6 h/day for 20 d (120000 ppm hours) showed decreased activity and bone marrow hypoplasia (Inoue, 1975).

It would appear that the chronic effects of toluene are more concentration than time dependent. It would also appear that the effects are noted first in the central nervous system and then, at somewhat higher concentrations, in the liver, observations which were also noted upon intraperitoneal and subcutaneous injection of the material (Ungvary et al., 1976).

3.4.3. MUTAGENICITY AND GENOTOXICITY

There were no reports of the mutagenicity of toluene in bacterial systems although these results are difficult to interpret as the material is highly toxic to bacteria. (NRC, 1981). Toluene has been reported to produce chromosome damage in rats after injection at 1 g/kg (Lyapkalo, 1973) or inhalation of vapour at 50 ppm for an unspecified time (Dobrokhotov and Enikeev, 1977).

3.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Increased skeletal abnormalities and retardation of fetal development were reported in mice exposed to 1600 ppm toluene (24 h/day from day 6 to 13 of gestation (NRC, 1981). Pregnant rats exposed to toluene at 2000 ppm for an unspecified period reportedly showed embryo toxicity but no teratogenicity (Hudak et al., 1977).

3.4.5. CARCINOGENICITY

Single incidences of a skin papilloma and a skin carcinoma were reported after topical treatment of a total of 30 mice with 10 to 20 uL of toluene 2 times a week for 72 weeks (Lijinski and Garcia, 1972) however these are difficult to interpret in the absence of control data or dose response. Poll (1963) failed to demonstrate carcinogenicity attributable to toluene in mice following topical application to the skin 3 d/week for a lifetime. The available evidence does not suggest that toluene is carcinogenic.

3.5. HUMAN TOXICOLOGY

Most reports of human exposure to toluene do not describe clinical or physiological responses to exposure under occupational conditions (100-400 ppm NAS, 1977). Inhaled toluene is excreted primarily as hippuric acid or benzoic acid in man (NAS, 1977). Effects reported in humans do include adverse mental changes, altered psychomotor ability, irritability, disorientation and unconsciousness after high exposure (Hayden et al., 1977, Weisenberger, 1977 and Matsushita et al., 1975) Occupational exposure to levels in the region of 200 ppm for 15 years were not reported to increase chromosomal aberrations in lymphocytes (NRC, 1981).

3.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

Recommended time weighted average US TLV, PEL and TWA levels are 100, 200 and 200 ppm respectively and the action level is 50 ppm (NIOSH/OSHA, 1981) while the lowest TWA concentration level is that of 15 ppm in the USSR. (ILO, 1980) NAS (1980) has suggested the following suggested no-adverse response levels (SNARLs) for drinking water:

24 h exposure	420 mg/L
7 d exposure	35
Chronic exposure	0.34

The chronic SNARL is based on a 20% intake from water and the NOEL of 300 ppm in Fisher rats (CIIT, 1978)

3.6. HAZARD ASSESSMENT

Concentrations of toluene in Edmonton water were normally reported at less than the detection limits of 0.1 ug/L (0.1 ppb), however, in a few instances levels as high as 1.0 ug/L were reported. Since these are well below the chronic SNARL developed by the NAS, the hazard from chronic exposure to toluene from Edmonton drinking water is judged to be negligible, even in the worst case situation.

4. NAME: XYLENE

4.1. CAS No.: 108-38-3

4.2. SOURCES AND USES

Xylenes are formed during the refining of petroleum and are produced during the distillation of coal tar and coal gas. Xylenes are used as solvents in the preparation of cements and protective coatings and in the manufacture of organic chemicals. Although a number of isomers (o, m and p) of xylenes exist, the m isomer predominates in commercially used xylene. Xylene has been reported from the finished drinking water of one US city at a concentration of 4.1 ug/L (NAS, 1979).

4.3. PHARMACOLOGY AND METABOLISM

Exposure of humans to a 1:1:1 mixture of o-, m- and p-xylene at concentrations of 0.2 to 0.4 mg/L in air demonstrated that about 65% of inspired xylene was retained by the lungs. After exposure, about 5% of the retained xylenes were excreted unchanged via the lungs and about 95% was excreted in the form of methylhippuric acids or, to some extent, as xylenol (Sedivec and Flek, 1976). Xylene may also be absorbed through the skin in humans (Engstrom et al., 1977) after which it undergoes metabolism in a similar manner to that observed after inhalation exposure.

Oral exposure of rats to xylenes at doses of 100 mg/kg body weight resulted in metabolism to the following (NAS, 1979):

o-xylene - 3,4-dimethylphenol, 2-methylbenzyl alcohol and 2,3-dimethylphenol.

m-xylene - 2,4-dimethylphenol

p-xylene - 2,5-dimethylphenol

Incubation of p-xylene with rat liver or lung microsomes was reported to result in the formation of p-methylbenzyl alcohol (Harper et al., 1974).

4.4. ANIMAL TOXICOLOGY

4.4.1. ACUTE

Xylenes have relatively low toxicity and have initial symptoms of exposure in humans related to irritation of the respiratory epithelium and in the case of high levels, central nervous system depression. Selected toxicity data is presented in the table below.

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
o-, *m*- and *p*-xylene	Rat	?	Oral	LD_{50}	5000	Miller, 1976
Mixed		?			4300	Patty, 1981
Mixed	Rat	?	Inhl 4h	LC_{50}	6350 ppm	Patty, 1981
Mixed	Rat	?	IP	LD_{50}	1500-2000	Miller, 1976
o-xylene		?	SC	LD_{50}	2500	Miller, 1976
m- and *p*-			SC	LD_{50}	5000	Miller, 1976
m-xylene	Mouse	?	Inhl 6h	LC_{50}	5267	EC, 1984

Acute exposures to xylenes have also been reported to increase blood enzyme levels associated with impairment of liver function in rats (Patel *et al.*, 1976) and guinea pigs (Divincenzo and Krasavage 1974).

4.4.2. CHRONIC

A number of effects resulting from chronic exposure to xylenes have been reported in the literature. Decreased red- and white-blood cell counts have been reported in rabbits exposed via inhalation to xylene at 1150 ppm for 40 to 55 days (NAS, 1979). Carpenter *et al.* (1975) reported neither histological nor haematological effects in rats and dogs exposed to 175, 460 and 805 ppm o-xylene for 6 h/day, 5 days per week for 13 weeks.

4.4.3. MUTAGENICITY AND GENOTOXICITY

Xylenes have been reported as being non-mutagenic in Salmonella typhimurium and Saccharomyces cerevisiae mutation assays either with or without activation by the S9 liver microsomal fraction (EC, 1984). Xylenes have not been reported to cause chromosome aberrations in bone marrow cells from rats exposed to concentrations of 0.004 to 0.441 mg/L in air (EC, 1984).

4.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Xylenes have been reported to cause developmental defects in developing chicken eggs (NAS, 1979) at unspecified concentrations. Evidence of teratogenicity was reported in rats exposed to 1000 mg/m^3 for 24 h/day from day 1 to 21 of gestation, however, exposure to concentrations of 435 and 1700 mg/m^3 for 6 h/day from day 6 to 15 of gestation did not result in terata or other signs of fetal toxicity (EC, 1984). This suggests the possible saturation of detoxification mechanisms in the rats exposed for 24 h/day and resultant maternal toxicity.

4.4.5. CARCINOGENICITY

No studies on the carcinogenic potential of xylenes were found in the literature.

4.5. HUMAN TOXICOLOGY

Symptoms of exposure in humans include eye irritation, severe lung congestion, impairment of renal function and minimal liver damage (these in the case exposure to 10000 ppm) (NAS, 1979). Exposure to levels of 100 to 200 ppm in air have been reported to result in slight impairment of equilibrium which could be counteracted by light exercise (EC, 1984). Long-term exposure to high levels in occupational settings has been associated with clinical signs of liver enlargement and nephrosis (Patty, 1981).

4.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

The TLV for xylenes has been established at 100 ppm (435 mg/m^3) for 8 h exposure in an occupational setting.

Based on acute and subchronic exposures to xylenes, NAS (NAS, 1980) calculated the following suggested no adverse response levels (SNARLs) for drinking water but were unable to calculate a chronic SNARL for lack of adequate data.

24 h exposure	21 mg/L
7 d exposure	11.2

4.6 HAZARD ASSESSMENT

The levels of m- and p-xylene reported from Edmonton drinking water were usually less than the detection limits of 0.1 ppb (0.1 ug/L) although there were occasional reports of levels as high as 12 ug/L for m-xylene. On the basis of a worst case of a 60 kg person consuming 2 L of water per day, this would be equivalent to about 0.37 ug/kg body weight/day. This is some 30000-fold less than the 7 day chronic SNARL. On the basis of the limited data, the human health hazard from exposure to xylenes via Edmonton drinking water appears negligible, however, concern is expressed at the lack of adequate chronic toxicity data.

5. NAME: ETHYLBENZENE

5.1. CAS No.: 100-41-4

5.2. SOURCES AND USES

Ethylbenzene is used as a solvent in paints, rubber and in the chemical industry. It is also used as a feed stock in the production of styrene and, as such, is an important industrial chemical.

5.3. PHARMACOLOGY AND METABOLISM

Ethylbenzene is absorbed via the lungs, skin and intestinal tract. Efficiency of uptake from lungs has been reported in the range of 49 to 66% in men exposed to concentrations in the range of 4 to 40 ppm (Bardodej and Bardodejova, 1970; Angerer and Lehnert, 1979). Absorption via the skin from either air or contaminated water was reported to be minimal (Gromiec and Piotrowski, 1984) although it did occur.

Ethylbenzene was metabolized in workers exposed at occupational levels to a number of metabolites such as 2-ethylphenol (Angerer and Lehnert, 1979) phenyl glyoxylic acid and mandelic acid (Bardodej and Bardodejova, 1970). Although ethylbenzene may be excreted from the body via the lungs it would appear that this route of excretion is relatively minor. Chin et al. (1980) reported that between 80 and 90% of ethylbenzene absorbed after inhalation exposure in dogs was excreted in the urine. The high lipid solubility of ethylbenzene probably accounts for its tendency to distribute into the lipid rich organs in the body (Gerarde, 1959) and for its slower rate of dissipation compared to benzene and toluene.

5.4. ANIMAL TOXICOLOGY

5.4.1. ACUTE

In general, ethylbenzene is of low acute toxicity in man and animals where the major symptom of exposure is depression of the central nervous system. Representative acute toxicities are given below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Ethylbenzene	Rat	?	Oral	LD_{50}	4940	EPA, 1980
	Rat	?	Inhl(4h)	LC_{50}	4000 ppm	EPA, 1980
	Rabbit	?	Dermal	LD50	17.8 ml/kg	EPA, 1980
	Man	M	Oral	LD_{50}	5000	Driesbach, 1980

5.4.2. CHRONIC

Dietary exposure of rats to ethylbenzene over a 6 month period resulted in dose related increases in weight of organs such as liver and kidney at doses above 136 mg/kg body weight/day. Histopathological changes were noted in the liver and kidney but these were not associated with tumour formation. No effects on hematopoiesis were seen (Wolf et al., 1956).

Inhalation studies have been conducted in rats, guinea pigs and in monkeys (Wolf et al., 1956) but failed to show carcinogenic effects. In rats exposed to ethylbenzene at 2200 ppm (7 h/day for 103 exposures), a moderate kidney weight increase and depression of growth was reported. These effects were also noted at lower doses but were less severe. In guinea pigs, exposure to 1250 ppm (7 h/day for 138 exposures) resulted in a slight depression in growth rate while at 600 ppm (7 h/day for 130 exposures) slight histopathology of the testes was reported. Exposure of Rhesus monkeys to 600 ppm (7 h/day for 130 exposures) resulted in slight histopathology of the testes but exposure of both monkeys and guinea pigs to 400 ppm (7 h/day for 130 exposures) was reported to be without effect.

5.4.3. MUTAGENICITY AND GENOTOXICITY

No evidence for the genotoxicity of ethylbenzene has been reported in the literature although assays have been conducted using bacterial systems such as the Ames' test (Salmonella typhimurium, with and without activation) and the yeast test (Saccharomyces cerevisiae) (Dean et al., 1985) Chromosome aberrations were not reported in rat liver cells in culture when exposed to concentrations as high as half that required to inhibit growth by 50%. No data on human studies has been reported in the literature.

5.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Teratogenicity has been studied in the rat and the rabbit (Andre et al., 1981). In the rat studies, animals were exposed to levels up to 1000 ppm (7 h/day for 5 exposures per week) for three weeks prior to mating. After mating, the animals were exposed using the same regimen up to day 19 of gestation. Maternal and paternal toxicity was evident in increased liver, kidney and spleen weights but there was no evidence of teratogenicity or fetotoxicity in the offspring. Mating frequency was apparently reduced in the exposed groups although this is probably a behavioral effect of the exposure rather than a toxic endpoint.

Similar studies on rabbits exposed to levels up to 1000 ppm (7 h/day for 5 exposures per week) for three weeks prior to mating and for 30 days after mating did not indicate any teratogenic effects although liver weights increased in the high dose group (Andre et al., 1981).

5.4.5. CARCINOGENICITY

No specific carcinogenicity data have been reported in the literature although tumors were not observed in the chronic exposure studies reported above.

5.5. HUMAN TOXICOLOGY

Driesbach (1980) reported an LD_{50} of 5g/kg body weight in man, however, the major response to exposure to vapours of ethylbenzene in man is irritation of the eyes, nose and throat (1000-5000 ppm). Dizziness and central nervous system effects have also been reported at the higher concentrations in this range while irritation, fatigue and headache have been reported at concentrations in the range of 100 ppm. (EC, 1984).

5.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

Recommended time weighted average US TLV and PEL levels are 100, 100 ppm respectively and the action level is 50 ppm (EC, 1984). The lowest MAC concentration level is that of 25 ppm in Poland.

5.6. HAZARD ASSESSMENT

In the absence of indications of mutagenicity, teratogenicity or carcinogenicity it is appropriate to assess the hazard of ethylbenzene on the basis of chronic toxicity. The levels of ethylbenzene usually reported in Edmonton drinking water were less than the detection level of 0.1 ug/L (ppb) although occasional levels up to 1.5 ug/L were reported. On the basis of a 60 kg person consuming 2 L of water a day, worst case daily dose based on consumption of water containing 1.5 ug/L would be 0.05 ug/kg body weight/day. This is some 2.6 million fold less than the lowest daily dose reported to cause effects in laboratory rats, suggesting that exposure to ethylbenzene via Edmonton drinking water is a negligible human health risk.

6. NAME: PHENYLACETALDEHYDE

6.1. CAS No.: 122-78-1

6.2. SOURCES AND USES

Phenylacetaldehyde is a naturally occurring substance which has been reported from a number of essential oils such as neroli, narcissus, magnolia, lily, rose, tea and a number of citrus species (Opdyke, 1979). It has also been reported as present in the oil of *Nepeta nepetella*, a species of plant related to catnip (Bicchi *et al.*, 1984) and is a metabolic product found in the fungus *Botrytus cinerea* where, in conjunction with other naturally produced compounds, it is thought to contribute to the musty odour sometimes detected in public water supplies (Kikuchi *et al.*, 1983). It has also been reported as a constituent of the attractant for the Queensland fruit fly, *Dacus tyroni* (Morton and Bateman, 1981) and for the cabbage looper *Trichoplusia ni* (Opdyke, 1979).

6.3. PHARMACOLOGY AND METABOLISM

While little specific information is available on the pharmacokinetics of phenylacetaldehyde, it appears to be readily taken up by the gastrointestinal tract and metabolized by dehydrogenase enzymes in the liver to phenylacetic acid (Panchenko *et al.*, 1985).

6.4. ANIMAL TOXICOLOGY

6.4.1. ACUTE

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
phenyl-acetaldehyde	Rat	?	Oral	LD_{50}	3890	Opdyke, 1979
	Mouse	?	Oral	LD_{50}	3890	Opdyke, 1979
	Guinea pig.	?	Oral	LD_{50}	3890	Opdyke, 1979

6.4.2. CHRONIC

No information was available in the literature.

6.4.3. MUTAGENICITY AND GENOTOXICITY

No information was available in the literature.

6.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No information was available in the literature.

6.4.5. CARCINOGENICITY

No information was available in the literature.

6.5. HUMAN TOXICOLOGY

No information was available in the literature.

6.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

This material is classified as "generally recognized as safe" (GRAS) by FEMA (Opdyke, 1979) and has an ADI of 0 to 1 mg/kg body weight/day.

6.6. HAZARD ASSESSMENT

Because this material is generally recognized as safe, with an ADI of 1 mg/kg body weight/day the levels reported in Edmonton drinking water (<10 ppb) do not present a human health hazard.

7. NAME: PHENYLACETIC ACID

7.1. CAS No.: 122-78-1

7.2. SOURCES AND USES

Phenylacetic acid (also known as bezeneacetic acid) is used in the manufacture of a number of industrial chemicals such as perfume, drugs, plant hormones, fungicides and flavorings (NAS, 1979). Phenylacetic acid has also been reported from a number of natural sources such as the urine of black-tailed deer (*Odocoileus hemionus columbianus*) (Crump *et al.*, 1984) and from the scales of the corn earworm (*Heliothis zea*) (Gueldner *et al.*, 1984). Durden and Boulton (1982) reported normal levels of phenylacetic acid in the brains of rats to be of the order of 30 ng/g.

7.3. PHARMACOLOGY AND METABOLISM

Phenylacetic acid is rapidly absorbed through the tissues of the throat (NAS, 1979) and presumably other membranes in the gastrointestinal tract. Potempska *et al.* (1984) reported that phenylacetic acid was the major metabolic product of the amino acid, phenylalanine in persons suffering from phenylketonuria. They have also shown that phenylacetic acid (in the form phenyl-acetyl-CoA) inhibits the activity of the enzyme choline acetyltransferase *in vitro*, causing complete inhibition of synthesis at concentrations of 10^{-4} M. Choline acetyltransferase is one of the enzymes responsible for the synthesis of acetylcholine, an essential neurotransmitter in man and other animals. Although phenylacetic acid inhibits this enzyme, it is important to note that it is a metabolite of a naturally occurring amino acid and is a normal, endogenous constituent of the body in man and animals. While excessive concentrations may be hazardous, exposures which result in little increase in endogenous levels are unlikely to be a significant hazard. Phenylacetic acid has also been reported to inhibit phototropism in corn (*Zea mays*) seedlings with an I_{50} of about 10^{-1} M (Vierstra and Poff, 1981). Garcha et al. (1985) reported relatively rapid dissipation of phenylacetic acid from the blood of cats without apparent accumulation.

7.4. ANIMAL TOXICOLOGY

7.4.1. ACUTE

The oral LD_{50} of phenylacetic acid in rats was reported as 1.63 g/kg body weight and the IP LD_{50} in mice was less than 300 mg/kg body weight (NAS, 1979). No other reports of acute toxicity were found in the literature.

7.4.2. CHRONIC

Treatment of suckling rats with phenylacetic acid has been reported to lead to morphological and biochemical changes in the brain (Wen et al., 1980 and Loo et al., 1980) as well as alterations in behaviour (Fulton et al., 1980).

7.4.3. MUTAGENICITY AND GENOTOXICITY

No information was found in the literature.

7.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

It has been reported that, at doses 3.2 mg/kg body weight administered on the 12th day of gestation, phenylacetic acid caused changes in body weight, retarded skeletal ossification and resulted in resorption of embryos at twice the rate of control animals (NAS, 1979).

7.4.5. CARCINOGENICITY

It has been reported that phenylacetic acid is not carcinogenic in rabbits when given over a period of 40 days by either IV or SC routes at unspecified doses (NAS, 1979).

7.5. HUMAN TOXICOLOGY

No information was found in the literature.

7.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No information was found in the literature.

7.6. HAZARD ASSESSMENT

Phenylacetic acid was reported from Edmonton water at levels of less than 20 ppb (ug/L). Since the toxicological data is insufficient for an unequivocal hazard assessment, it is relevant to compare intake to levels which may naturally be present in the body. Given a worst-case of 20 ug/L, and a level of water consumption of 2 L/day in a 60 kg person, the daily dose would be 0.66 ug/kg/day. This is some 45 fold less than the endogenous concentration of phenylacetic acid in the brain (and presumably other tissues) and, on the basis of redundance in the system, it is unlikely to have adverse health effects. On the basis of limited toxicological evidence, the levels of phenylacetic acid in Edmonton water are unlikely to present a significant health risk.

8. NAME: BENZENEACETONITRILE

8.1. CAS No.: 140-29-4

8.2. SOURCES AND USES

Benzeneacetonitrile is thought to be an odorous metabolite of blue-green algae. Slater and Blok (1983) suggested that this metabolite contributes to the "muddy" or "musty" taste and odour of water in Buffalo Pound Lake in Southern Saskatchewan. As well, benzeneacetonitrile has been identified as a natural component of garden cress, sugar beet and other plants. Benzeneacetonitrile has several auxin-like properties. Work with sugar beet plants suggest that benzeneacetonitrile may be an inactive, reserve auxin which could be converted to the growth-active benzeneacetic acid when required for growth regulation. This circumvents accumulation of toxic levels of benzeneacetic acid (Wheeler et al, 1980). Benzeneacetonitrile was not found to have significant industrial uses.

8.3. PHARMACOLOGY AND METABOLISM

In mammals, oxidative metabolism of benzeneacetonitrile by the hepatic microsomal enzyme system leads to cyanide liberation (Tanii and Hashimoto, 1985). The toxicity of benzeneacetonitrile is thought to be mediated by the cyanide ion. Significant accumulation of cyanide can lead to inhibition of metal-containing enzymes, most notably cytochrome oxidase, as well as inactivation of methemoglobin. Detoxication of cyanide occurs via conversion to thiocyanate by the enzyme rhodanase. Thiocyanate is subsequently excreted in the urine. Low concentrations of cyanide (0 to 10.7 ug CN^-/100 ml) are frequently found in normal human blood (Feldstein and Klendshoj, 1954).

8.4. ANIMAL TOXICOLOGY

8.4.1. ACUTE

The oral LD_{50} for male ddY mice administered benzeneacetonitrile was reported as 45.52 mg/kg body weight. Pretreatment with carbon tetrachloride, a hepatic mixed function oxygenase system inhibitor, significantly decreased acute toxicity (Tanii and Hashimoto, 1985). No further data were found in the literature.

8.4.2. CHRONIC

No data were found in the literature.

8.4.3. MUTAGENICITY AND GENOTOXICITY

Benzeneacetonitrile was found to be non-mutagenic in the Ames *Salmonella typhimurium* assay using the strains TA98, TA100, TA1535, and TA1537 with and without the S-9 from aroclor-induced rats (Florin *et al.*, 1980)

8.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No data were available in the literature.

8.4.5. CARCINOGENICITY

No data were available in the literature.

8.5. HUMAN TOXICOLOGY

No data were found in the literature.

8.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No data were found in the literature.

8.6. HAZARD ASSESSMENT

Any human health hazard resulting from exposure to benzeneacetonitrile would most likely be due to its highly toxic metabolite, cyanide. An acceptable daily intake (ADI) of 0 to 8.4 mg/person/day of cyanide has been calculated for man (WHO, 1984). The levels of benzeneacetonitrile found in Edmonton drinking water were approximately 1 ug/l. Assuming consumption of 2 litres per day and maximum conversion to cyanide, this would correspond to 0.44 ug cyanide intake per day. This value is 10000-fold less than the ADI of 8.4 mg cyanide. This level would be equivalent to 1.3 million-fold less than the acute LD_{50} in mice. Based on the above calculations, benzeneacetonitrile levels in Edmonton drinking water do not appear to present a human health hazard. An unequivocal assessment, however, cannot be made due to a lack of chronic toxicological data.

9. NAME: PHENYLPROPIONIC ACID

9.1. CAS No.: 501-52-0

9.2. SOURCES AND USES

Phenylpropionic acid has no industrial uses but could be formed as an oxidation product of n-propylbenzene. n-Propylbenzene is a petroleum byproduct which is also produced during the manufacture of isopropylbenzene (cumene). It is used in the preparation of dyes and the manufacture of methylstyrene (NAS, 1979). It has been reported from the water of two US cities at concentrations of 0.01 and 0.05 ug/L (NAS, 1979). Phenylpropionic acid has not been reported from water by NAS. (NAS, 1977, 1979, 1982, 1983).

9.3. PHARMACOLOGY AND METABOLISM

No specific information on the metabolism of phenylpropionic acid was found in the literature, however, n-Propylbenzene is readily absorbed from the gastrointestinal tract (NAS, 1979). Metabolism of n-propylbenzene in rats is via side-chain oxidation as well as ring hydroxylation (Gerarde and Ahlstrom, 1966) with the former the preferred route. This metabolism leads to the formation of, amongst others, phenylpropionic acid and then via beta oxidation, to benzoic acid which is also subject to review in this document.

9.4. ANIMAL TOXICOLOGY

9.4.1. ACUTE

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
phenyl-propionic acid	No information available					
n-propyl-benzene	Rat	?	Oral	LD_{LO}	4830	Patty, 1981
		?	Oral	LD_{20}	5000	"
			Oral	LD_{50}	7500	(NAS, 1979)

9.4.2. CHRONIC

No information was available on phenylpropionic acid, however, a six month oral chronic study on n-propylbenzene conducted in rabbits (Gerarde and Ahlstrom, 1966) at doses of 0.25 and 2.5 mg/kg body weight/day showed no indications of toxicity except for mild protein dystrophy in some of the animals.

9.4.3. MUTAGENICITY AND GENOTOXICITY

No data were found in the literature.

9.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No data were found in the literature.

9.4.5. CARCINOGENICITY

No data were found in the literature.

9.5. HUMAN TOXICOLOGY

No data were found in the literature.

9.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No occupational exposure limits have been established for phenylpropionic acid or n-propylbenzene.

9.6. HAZARD ASSESSMENT

No assessment of phenylpropionic acid has been attempted in the literature. NAS (1979) were unable to assess health hazard of n-propylbenzene because of a lack of data. Levels of phenylpropionic acid reported in Edmonton drinking water were less than the detection limits of 20 ug/L (20 ppb). As an organic acid, it would be relatively easily excreted from the body and, if metabolized, would lead to the formation of benzoic acid. The latter compound is a food additive and food is likely to present a much more significant source than water. The paucity of toxicological data does not allow a full health hazard assessment, however, the limited information available does not suggest that this chemical presents a health hazard via drinking water.

10. NAME: CHLOROBENZENE

10.1. CAS No.: 108-90-7

10.2. SOURCES AND USES

Mono-chlorobenzene is used in the manufacture of pesticides, aniline, phenol and aniline as well as an intermediate in the manufacture of dyestuffs. It may also be formed during the chlorination of water and was reported from water supplies in a number of US cities in concentrations ranging from 4.7 to 5.6 ug/L (NAS, 1977) as well as from the water supplies of several Canadian cities (Otson _et_ _al_., 1982).

10.3. PHARMACOLOGY AND METABOLISM

Chlorobenzene is absorbed from the gut via inhalation and through the skin. The liver is the primary site of toxicity and the toxic effect is thought to be due to the formation of a toxic intermediate by the monooxygenase enzyme system of the liver. Intraperitoneal injection of this material caused signs of liver toxicity in male mice (Shelton and Weber, 1981) but this effect was not observed in rats (Yang _et_ _al_., 1979) although an increase in bile flow was measured. S-(p-chlorophenyl)mercapturic acid was reported as a metabolite of chlorophenol excreted in the urine of animals (Reid _et_ _al_., 1971).

10.4. ANIMAL TOXICOLOGY

10.4.1. ACUTE

Chlorobenzene has been reported to have an acute oral LD_{50} of 2910 mg/kg body weight in rats (NAS, 1977) and an acute inhalation LC_{50} of 8000 to 9000 mg/m^3 in female mice (Bonnet _et_ _al_., 1979).

10.4.2. CHRONIC

A no-effect dose in rats of 0.001 mg/kg/day over a 7 month period of exposure has been reported (NAS, 1977) while dogs are reported to show no effect doses of 54 mg/kg in single exposures (NAS, 1977). Feeding of chlorobenzene to rats at doses of 200, 400 and 800 mg/kg/day for 14 days caused decreases in levels of cytochrome P450 in the liver at the high dose and increases in glucuronyl transferase at all doses (Carlson and Tardiff, 1976). Similar effects were noted by Ariyoshi et al. (1981) in male Wistar rats following intraperitoneal injection.

10.4.3. MUTAGENICITY AND GENOTOXICITY

Reports on the genotoxicity of this material are few in the literature but, where studied, the material has been reported as non-mutagenic (Lyon, 1976 and Lawlor et al., 1979).

10.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Reports on the reproductive and teratology of this material were not found in the literature.

10.4.5. CARCINOGENICITY

Chlorobenzene was tested for carcinogenic potential in Fisher rats and $B6C3F_1$ mice using gavage of the material dissolved in corn oil (NTP, 1982c). Animals were dosed with 0, 60 and 120 mg/kg body weight, five days per week for two years in the case of the rats and female mice and at 30 and 60 mg/kg body weight in the case of male mice. A dose related increase in hepatic neoplastic nodules was reported in the rats (2/50; 4/49 and 8/49 at 0, 60 and

120 mg/kg body weight respectively) and was stated to indicate some, but not clear evidence of carcinogenicity in male rats (NTP, 1982c). In their evaluation of the evidence of liver carcinogenicity in rats, NAS (1983) calculated that the estimated human lifetime risk of cancer at a daily dose in water of 1 ug/L was 3.71 x 10^{-8} and that the upper 95% confidence estimate of lifetime human cancer risk was 2.13 x 10^{-7} per ug/L in water. This calculation was based on linear extrapolation of data corrected for surface area and dosing regimen but with no consideration for threshold or species differences in terms of susceptibility. The committee in fact stated (NAS 1983) that the calculation was based on limited evidence.

10.5. HUMAN TOXICOLOGY

Mono-chlorobenzene has been reported as an irritant of the upper respiratory tract and appears to act as a central nervous system depressant. An early epidemiological study (Rozenbaum et al., 1947) reported an excess of nervous system abnormalities in factory workers exposed to this compound in the workplace. The levels of exposure in this situation are likely so much higher than those in water that these observations are of little significance. The USSR has suggested that the maximum permissable concentration in drinking water should be 20 ug/L (NAS, 1977).

10.6. HAZARD ASSESSMENT

Levels of chlorobenzene in Edmonton water were consistently reported to be less than the detection threshold of 0.2 ug/L (ppb). This level is below the guideline level for water in the USSR. On the basis for some concern regarding the carcinogenicity of this compound and using the above extrapolation of cancer risk, a worst case level in Edmonton water would suggest an upper risk of 4.26 x 10^{-8} of developing cancer. Since it is generally considered that a 1 x 10^{-6} risk may be regarded as negligible, there is little concern that levels of chlorobenzene in Edmonton water represent a possible human health risk.

11. NAME: BENZOPHENONE (DIPHENYL METHANONE)

11.1. CAS No.: 119-61-9

11.2. SOURCES AND USES

Benzophenone is prepared by the Friedel-Crafts ketone synthesis method using benzene and benzoyl chloride. Benzophenone has a persistant, delicate, rose-like aroma and is used as a fixative for heavy perfumes and soaps. As well, benzophenone is used in the manufacture of antihistamines, hypnotics, and insecticides. Benzophenone is a semi-volitile constituent of cigarette smoke condensate (Windholz et al, 1983).

11.3. PHARMACOLOGY AND METABOLISM

At lethal doses benzophenone produces sedation, progressive depression of motor activity, unstable gait, tremors and respiratory impairment (Caprino et al, 1976).

11.4. ANIMAL TOXICOLOGY

11.4.1. ACUTE TOXICOLOGY

There is limited data in the literature on the acute toxicity of benzophenone. Available data is summarized below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
benzophenone	mouse	?	i.p.	LD_{50}	727	Caprino *et al*, 1976
	mouse	?	oral	LD_{50}	2895	Caprino *et al*, 1976

11.4.2. CHRONIC

No data were available in the literature.

11.4.3. MUTAGENICITY AND GENOTOXICITY

No data were available in the literature.

11.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No data were available in the literature.

11.4.5. CARCINOGENICITY

Stenback and Shubik (1974) examined the potential carcinogenicity of benzophenone in female Swiss mice by administering repeated applications of the chemical (0.02 ml of either of 5% or 25% concentration) on the skin for the life-span of the animals. A statistically significant increase in tumor incidence caused by the chemical treatment was not seen.

11.5. HUMAN TOXICOLOGY

Ingestion of benzophenone causes gastrointestinal disturbances. Contact causes eye irriation and, if prolonged, irritation of skin (Weiss, 1980).

11.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No data were available in the literature.

11.6. HAZARD ASSESSMENT

There is insufficient data for a complete hazard assessment of benzophenone. Levels of this compound in Edmonton drinking water did not exceed the detection limits of 10 ppb (10ug/L). The limited data suggests that the material is not a carcinogen

12. NAME: TETRAHYDROMETHYLBENZOFURANONE

12.1. CAS No.: 15356-74-8
81800-41-1

12.2. SOURCES AND USES

Tetrahydromethylbenzofuranone has been identified as a natural constituent of rose oil (Liu et al, 1982).

12.3. PHARMACOLOGY AND METABOLISM

No data were found in the literature.

12.4. ANIMAL TOXICOLOGY

No data were found in the literature.

12.4.1. ACUTE

No data were found in the literature.

12.4.2. CHRONIC

No data were found in the literature.

12.4.3. MUTAGENICITY AND GENOTOXICITY

No data were found in the literature.

12.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No data were found in the literature.

12.4.5. CARCINOGENICITY

No data were found in the literature

12.5. HUMAN TOXICOLOGY

No data were found in the literature.

12.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No data were found in the literature.

12.6. HAZARD ASSESSMENT

Due to the paucity of data on tetrahydrotrimethylbenzofuranone no hazard assessment can be made.

13. NAME: METHYLENE CHLORIDE

13.1. CAS No.: 75-09-2

13.2. SOURCES AND USES

Methylene chloride is used in the manufacture of a number of consumer products such as paints, paint removers, solvents, cleaners and as a component of aerosol and other pressurized spray formulations. It is also used in the food industry as an extractant for decaffeination of coffee and tea and in the preparation of spice and hop extracts. Methylene chloride has been found in treated water but is rarely found in raw water. It was reported in the supplies of several US cities at concentrations of <1 ug/L and the highest concentration reported was 1.6 ug/L (NAS, 1977).

13.3. PHARMACOLOGY AND METABOLISM

In the case of methylene chloride vapour, the majority is absorbed via the lungs and becomes fairly evenly distributed throughout the body (NAS, 1978). Intraperitoneal injection of 412 to 930 mg/kg body weight methylene chloride in SD rats showed that ca. 90% was eliminated from the lungs. Of the balance, 3% was exhaled as CO_2 and 2% as CO. A small quantity remained in the body (2%) and some (1%) was excreted as a metabolite in the urine (NAS, 1977). Rodkey and Collison (1977a) showed that injection of smaller doses of methylene chloride (17 mg/kg body weight) resulted in a larger fraction being metabolized and a total of about 75% was converted to CO_2 and CO. They were also able to show (Rodkey and Collison, 1977b) that conversion to CO was rate limited by enzyme saturation, an important observation as it suggests that the pharmacokinetics of this compound may change with the exposure concentration. This saturation has been observed to occur in rats and mice at concentrations of about 100 mg/kg body weight and at inhalation concentrations of 200 ppm in man and <500 ppm in rats (Kirschman, 1984).

This saturation of detoxication mechanisms is of particular importance in the interpretation of the carcinogenicity data below.

Withey (1984) and Angelo (1984) have demonstrated that methylene chloride is more readily absorbed from the gut when in water than in oil solution and that the rate of metabolic conversion is more rapid when methylene chloride is administered in water.

13.4. ANIMAL TOXICOLOGY

13.4.1. ACUTE

Methylene chloride has relatively low acute toxicity to animals. Selected reports of toxicity are given in the table below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Methylene	Rat	?	Oral	LD_{50}	1.6-2.3 mL/kg	NAS, 1977
		?	IP	LD_{50}	1.5 mL/kg	NAS, 1977
	Dog	?	IP	LD_{50}	0.95 mL/kg	NAS, 1977

Kimura _et_ _al_., (1971) reported that the first signs of intoxication in rats occurred at oral concentrations of 1 uL/kg body weight in rats.

13.4.2. CHRONIC

It has been reported (NAS, 1977) that rats allowed to drink water containing 125 mg/L of methylene chloride for 91 days showed no adverse effects. Likewise, a 6-month exposure of rabbits and dogs to 5000 ppm methylene chloride vapour caused no ill effects although a slight reduction in weight gain was observed (NAS, 1977). At vapour concentrations of 10000 ppm some injury was reported in guinea pigs exposed for 52 days. Long term exposure via drinking water has suggested no observable effect levels of 15 mg/kg body weight/day (EPA, 1981) and 5 mg/kg body weight/day (Hazelton Labs., 1981) in rats and 185 mg/kg body weight in mice (Hazelton Labs., 1983).

13.4.3. MUTAGENICITY AND GENOTOXICITY

Methylene chloride was reported to be negative in a *Drosophila* mutagenicity test (NAS, 1977) but Simmon (*et al.*, 1978) reported it to be mutagenic in a *Salmonella typhimurium* assay without activation. It has also been reported to be mutagenic in a yeast test (Callen *et al.*, 1980). In both systems, mutagenic activity followed a dose response confirming the significance of this observation in bacterial systems. Methylene chloride has not been reported to be mutagenic in mammalian systems.

13.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

It was reported that methylene chloride was neither teratogenic nor fetotoxic in rats and mice exposed to 1250 ppm for 7 h/day from days 6 to 15 of gestation (NAS, 1977). Hardin and Manson (1980) reported that exposure of rats to 4500 ppm before and during gestation did not result in any teratogenicity in offspring, however, some maternal toxicity was observed. Bornschein *et al.*, (1980) reported some behavioral effects in the progeny of rats exposed in the above studies although no physical effects were observed. The number of animals examined and the lack of standard criteria for evaluation of these effects make it difficult to assess the significance of these findings.

13.4.5. CARCINOGENICITY

Theiss *et al.*, (1977) reported a negative pulmonary tumour response in mice dosed with 160 to 800 mg/kg body weight for 16 days, however, a long-term inhalation study in rats indicated some evidence of carcinogenicity (Burek *et al.*, 1984). In this study (Burek *et al.*, 1984) rats and hamsters were exposed by inhalation to methylene chloride at concentrations of 0, 500, 1500 and 3500 ppm for 6 h per day, 5 days per week for 2 years. The female rats showed a dose-related increase in the total number of benign mammary tumors per tumour bearing rat at all doses. The male rats showed this response only at the two higher doses, numbers of tumour bearing rats were much lower and a clear dose response was not evident. In addition, the male rats also showed increases in the number of sarcomas associated with the salivary glands. The hamsters did not show these responses. In a second study (Nitschke et al., 1983) conducted in rats exposed to 0, 50, 200 and 500 ppm as above, a similar dose-response was observed in respect of benign mammary tumors in female rats.

In studies on mice, animals ($B6C3F_1$ strain) were exposed to methylene chloride at concentrations of 0, 60, 125, 185 and 250 mg/kg body weight in their drinking water. Lesions of the liver were observed in both the exposed and control mice to a similar extent. These lesions were more numerous in the exposed males than in the control males but no dose-response was observed. The no observable effect level for histopathological effects noted in this study was 185 mg/kg body weight/day (Hazelton Labs., 1983). Because of the route of exposure, this study is most relevant to the assessment of the potential carcinogenicity of methylene chloride in drinking water. The study showed no evidence of carcinogenicity. In this case one method of extrapolating risk is to assume a one-animal error in observation at the highest dose used to use this in the calculation of the maximum likelihood estimate of a dose equivalent to 10^{-6} lifetime risk of cancer. This proceedure yields a dose of 1.24 mg/kg body weight/day (Crump, personal communication).

The National Institute of Health inhalation study on the carcinogenicity of methylene chloride in mice ($B6C3F_1$ strain) and rats (F344/N) showed that there was "some evidence for carcinogenicity" for mammary tumors in male rats and "clear evidence of carcinogenicity" for tumors of the mammary gland in female rats and the lung and liver in male and female mice (NTP, 1986a). Rats and mice were exposed by inhalation to levels of 0, 1000, 2000 and 4000 ppm and mice were exposed to 0, 2000 and 4000 ppm for 6 h/day 5 days per week for 105 weeks. In both male and female rats a dose-dependent increase in benign mammary gland lesions (tumors) was observed, however, malignant mammary tumors did not show a dose response or an increase over control levels in either sex. Both alveolar/bronchial adenomas and carcinomas showed dose dependent increases in male and female mice and hepatocellular adenomas/carcinomas showed significant increases in the high exposure group in both male and female mice. From the point of view of assessing the risk associated with a drinking water route of exposure, this study is of lesser relevance, however, the most significant observation is that of bronchiolar/alveolar carcinoma in the female mice. The upper 95% confidence limit on the dose (calculated on the basis of 50% retention of inhaled methylene chloride) equivalent to a 10^{-6} risk of cancer is 0.17 mg/kg body weight/day.

13.5. HUMAN TOXICOLOGY

An epidemiological study of workers exposed to methylene chloride at concentrations of 50-100 ppm suggested no increases in diseases of the central nervous system, liver, gastrointestinal tract, cardiovascular system or the urinogenital tract (NAS, 1977). Winneke and Foder (1976) reported that acute exposure to 500 ppm methylene chloride in man resulted in a state of reduced activity and impairment of the central nervous system. Several recent epidemiological studies (Friedlander et al., 1978: Hearne and Friedlander, 1981 and Ott et al., 1983) have reported no association between exposure to methylene chloride in an occupational setting and incidence of cancer or other diseases. Although these studies were negative, exposure levels were low and they lacked sufficient statistical power to provide conclusive results.

13.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

The NIOSH TWA 10 hour day exposure level is 75 ppm (NAS, 1977) and the suggested no adverse response levels (SNARLs) for drinking water calculated from the acute animal toxicity data were as follows (NAS, 1980):

24 h exposure	35 mg/L
7 d exposure	5

Chronic exposure SNARLS could not be calculated for lack of data on chronic effects.

13.6. HAZARD ASSESSMENT

The observed carcinogenicity of methylene chloride in rats and mice is pivotal to the assessment of the hazard of this substance in man. The benign mammary tumors reported in rats are probably of lesser significance than the lung and liver tumors reported for mice as these tumors were site specific and not observed to be metastatic. The lung and liver tumors are significant, however, it should be noted that the exposure levels at which these were reported were far in excess of those at which saturation of the relevant enzyme systems would occur in the respective test animals (See above).

Levels of methylene chloride in Edmonton drinking water were reported to be less than the detection threshold of 10 ug/L (10 ppb) except in December of 1984 when a level of 23 ug/L was reported. In a 60 kg man consuming 2 L of water a day 10 ug/L represents a dose of 0.33 ug/kg body weight/day and the single observation of 23 ug/L represents a short-term exposure equivalent to a dose of 0.77 ug/kg body weight/day. Given that water was the sole source of intake, both these dose levels would be considerably less than the saturation dose for detoxification as mentioned above. Extrapolation of the two carcinogenicity studies suggest upper 95% confidence limits of the dose equivalent to a 10^{-6} lifetime risk of cancer of 1.2 mg/kg body weight/day for the drinking water study and 0.17 mg/kg body weight for the inhalation study. These levels are 1500 and 200 fold greater than the highest concentration of methylene chloride reported from Edmonton drinking water. On this basis the human health risk from exposure to these levels of methylene chloride via drinking water is negligible.

14. NAME: CHLOROFORM

14.1. CAS No.: 67-66-3

14.2. SOURCES AND USES

Chloroform, which enjoyed widespread use as an industrial chemical, drug and food additive in the past, currently has fewer uses. It is used as an industrial intermediate in the production of chlorofluoromethane refrigerants and as a laboratory chemical. In drinking water its major source is as a byproduct of the chlorination process and it is found in most water supplies where chlorination is used. It is usually found in higher concentrations where the levels of organic materials in water are high. Chloroform was found in the drinking water of almost all of the cities in the US at mean concentrations of 20 ug/L with the highest level being 311 ug/L (NAS, 1977). It is also present in the atmosphere at average levels of 14 parts per trillion (10^{-12} v/v) but levels in parts of North America have been reported as high as 31000 ppt (v/v)(EPA, 1985).

14.3. PHARMACOLOGY AND METABOLISM

Chloroform is easily absorbed via the lungs and the gastrointestinal tract and is distributed throughout the body with some tendency to concentrate in lipid-rich tissues (Cohen and Hood, 1969). In studies in humans (Fry et al., 1972) about half of an ingested dose of 7 mg/kg body weight of chloroform was expired unchanged from the lungs. The balance was metabolized to carbon dioxide. Other authors have reported similar findings, although there is considerable variation between individuals (Chiou, 1975 and Van Dyke et al., 1964). Metabolism of chloroform is catalyzed by the microsomal oxidase system leading to the formation of phosgene (Mansuy et al., 1977). Phosgene is highly reactive and is degraded via two pathways, either by reaction with water to give carbon dioxide and HCl (Brown et al., 1974) or by combination with tissue acceptors such as lipids or proteins (Docks and Krisna, 1976) but to a much lesser extent to nucleic acids (Uehleke et al., 1977). In mammals and man the principle site of metabolism is the liver but metabolism has also been reported in kidney, adipose tissue and muscle (Paul and Rubenstein, 1963; Butler, 1961). The degree of metabolism of chloroform in man is dependent on dose. At doses above 0.1 g some proportion of the dose is excreted unchanged via the lungs whereas, at doses less than this, all the chloroform is metabolized. This is of particular importance in the assessment of the effects of exposure to low doses such as would occur in consumption of water containing low levels of chloroform.

14.4. ANIMAL TOXICOLOGY

14.4.1. ACUTE

Summary data on the acute toxicity of chloroform is shown below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
chloroform	Rats	?	Oral	LD_{50}	2900	NAS, 1977
	Mice	?	Oral	LD_{50}	470- 120	NAS, 1977
	Dogs	?	Oral	LD_{50}	1450	NAS, 1977
	Mice	?	IP	LD_{50}	1740	NAS, 1977
	Man	?	Oral	Lethal dose	630	WHO, 1984

In man, acute exposure to chloroform results in depression of the central nervous system at non-lethal doses and was the basis for the extensive use of chloroform as an anesthetic.

14.4.2. CHRONIC

The major effect of chronic exposure to chloroform in mammals is damage to the liver. This effect is also observed as a non-lethal effect of acute exposure and has been reported from man (EPA, 1985). The target organs which normally show damage from acute toxicity to chloroform are the liver and the kidneys. Damage in the liver includes centrilobular necrosis, fatty degeneration and swelling (Groger and Grev, 1979). In the kidney, chloroform causes necrosis of the proximal and distal convoluted tubules (EPA, 1985). The mechanisms by which tissue damage occurs is through metabolism to reactive intermediates (phosgene) which bind to cell constituents and thus cause necrosis.

14.4.3. MUTAGENICITY AND GENOTOXICITY

EPA (1985) conducted an extensive review of the mutagenicity of chloroform and concluded that the majority of the assays yielded negative results. There was some evidence of reactivity of metabolites of chloroform with macromolecules which suggested that the negative results obtained in the bacterial and other in vitro mutagenesis assays may have been as a result of the very short life of these reactive intermediates.

14.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

The teratogenic and reproductive effects of chloroform have been examined in the rat, the mouse, and the rabbit.

Schwetz et al (1974a) exposed twenty female Sprague-Dawley rats to 30, 100, or 300 ppm chloroform (inhalation 7 hours/day) on days 6 through 15 of gestation. Evidence of maternal toxicity, including a decrease in the rate of maternal weight gain, was apparent at all dose levels. A decrease in food consumption during pregnancy was significant at the 100 and 300 ppm dose levels. A starvation control group was used at the 300 ppm dose level to differentiate between anorexic effects in the mother and chloroform-induced effects. Significant adverse effects, independent of those due to starvation in the mother, were noted at 300 ppm. These included: decreased fetal body weight, decreased conception rate, decreased number of live fetuses per litter, and an increased number of resorptions. Abnormalities of the skull and sternum and subcutaneous edema were also observed, but these were not statistically significant, possibly due to the small number of survivors in the 300 ppm group. An increased incidence of acaudia, missing ribs, and delayed ossification of sternebrae were observed in the 100 ppm group.

Murray et al (1979) exposed pregnant CF-1 mice to 100 ppm chloroform (inhaled) 7 h per day on gestation days 1-7, 6-15, or 8-15. The authors concluded that teratogenic (cleft palate) and embryotoxic effects, maternal toxicity and interference with pregnancy resulted from chloroform exposure.

Thompson et al (1974) examined the teratogenic effect of orally-administered chloroform on pregnant Sprague-Dawley rats and Dutch-Belted rabbits. Groups of 25 rats were intubated twice a day in divided doses of 20, 50, and 126 mg/kg body weight /day on days 6 through 15 of gestation. Groups of 15 rabbits were intubated once a day in doses of 20, 35, and 50 mg/kg body weight/day on days 6 through 18 of gestation. Rats receiving 50 and 126 mg/kgbody weight/day displayed signs of maternal toxicity, including decreased body weight gain, decreased food consumption, and fatty changes in the liver. No malformations were observed at any dose level. In rabbits, maternal toxicity was observed at the 50 mg/kg body weight/day level. In a range-finding portion of this study, Thompson et al (1974) noted signs of maternal toxicity, such as severe acute hepatitis and nephrosis, in both the rat and the rabbit at higher dose levels (rat, >126 mg/kg body weight/day; rabbit, >63 mg/kg body weight/day). The skeletal deformities and deficiencies in pregnancy maintenance reported by Schwetz et al (1974a) were not observed in this study.

Burkhalter and Balster (1979) studied the effect of chloroform in developing ICR mice. The parental generation was exposed by gavage to 31.1 mg/kg body weight/day 21 days prior to mating, during mating, until day 21 after birth. The offspring were exposed starting on day 7 until day 21 after birth. The authors concluded that 31.1 mg/kg body weight/day of chloroform produced no significant adverse behavioral effects in pups exposed both in utero and after birth. This conclusions must be considered tentative as one one dose and a small sample size was used.

After a review of the literature, EPA (1985) concluded that chloroform has the potential for causing adverse effects independent of those associated with maternal toxicity. These include a lowered rate of pregnancy maintenance, delays in fetal development, and production of terata in laboratory animals. Overall, the studies in which chloroform was administered by inhalation reported more severe outcomes than the intubation studies. EPA (1985) suggests that further studies be done to evaluate the importance of route of exposure.

14.4.5. CARCINOGENICITY

Several chronic studies have evaluated the carcinogenic potential of chloroform to a variety of laboratory animals. In all studies the route of administration was oral.

The National Cancer Institute (1976b) conducted a carcinogenesis bioassay on chloroform in male and female Osborne-Mendel rats and male and female $B6C3F_1$ mice. Chloroform was administered by gavage at two dose levels: the maximally tolerated dose and one half the maximally tolerated dose. For male rats this corresponded to 90 and 180 mg/kg body weight/day, for female rats 100 and 200 mg/kg body weight/day, 138 and 277 mg/kg body weight/day for male mice, and 238 and 477 mg/kg body weight/day for female mice. A statistically significant increase in the incidence of hepatocellular carcinomas in male and female $B6C3F_1$ mice was seen. At the high dose level, an increase in hepatocellular carcinoma of 98% in males and 95% in females was observed. At the low dose a 36% increase in males and an 80% increase in females was observed. Renal epithelial tumors (malignant and benign) were observed in male Osborne-Mendel rats. Female Osborne-Mendel rats did not have a carcinogenic response to chloroform in this study.

Jorgenson et al (1985) examined the carcinogenic potential of chloroform administered in drinking water, the route of exposure likely to be encountered by humans. The strains of mice and rats showing a positive response in the NCI (1976b) study were used. Chloroform was present in the drinking water of male Osborne-Mendel rats and female $B6C3F_1$ mice at concentrations of 0, 200, 400, 900, and 1800 mg/L. An increase in the incidence of renal tumors was seen in male Osborne-Mendel rats. The female B6C3F$_1$ mice, however, did not show an increase in liver tumors, as shown in the NCI (1976b) study. EPA (1985) suggested this lack of response may be related to the dosing regimen, absorption patterns, and peak blood and target tissue levels.

Palmer et al (1979) administered chloroform in toothpaste by gavage to male and female Sprague-Dawley rats for 80 weeks. Dose levels of 15, 75, and 165 mg/kg body weight/day were selected, based on the lowest toxic dose of 150 mg/kg body weight/day identified in a preliminary study. A carcinogenic response was not observed, but high rates of early mortality decreased the significance of these results.

The carcinogenicity of chloroform in toothpaste was evaluated in male and female beagle dogs by Heywood et al (1979). Chloroform was given orally in capsules 6 days per week for 7.5 years (0, 15 and 30 mg/kg/day). A small number of macroscopic and microscopic neoplasms were observed but no statistically significant carcinogenic effect was seen. EPA (1985), however, was uncertain as to whether 7 years was long enough for carcinogenicity testing with respect to the 13 to 14 year lifespan of the beagle dog.

A study by Capel et al (1979) showed that chloroform enhanced the growth of three types of murine tumors in mice. A dose of 15 mg/kg body weight/day chloroform administered for 14 days before or 14 days after inoculation of Ehrlich ascites tumour cells promoted significant growth and metastasis of the tumors.

In their Health Assessment Document for Chloroform, EPA(1985) concluded that there is sufficient evidence for the carcinogenicity of chloroform in experimental animals.

14.5. HUMAN TOXICOLOGY

Although no epidemiological cancer studies have dealt with chloroform per se, several studies have implicated an association between exposure to chlorinated drinking water and increased risks of bladder, colon, and rectal cancer (EPA, 1985). Chloroform, however, is a predominant component of chlorinated drinking water, particularly when the raw water is heavily organic-laden.

Cantor et al (1977) studied the association between cancer mortality rates and trihalomethane levels in municipal drinking water supplies in 76 countries, distinguishing between chloroform and non-chloroform components. The strongest correlation existed between bladder cancer mortality rates and trihalomethane levels, with brominated trihalomethanes more strongly correlated than chloroform. Correlations for chloroform were positive but nonsignificant.

Hogan et al (1979) conducted an ecological study of cancer rates based on NCI cancer mortality data for the years between 1950 and 1969 and on chloroform levels in finished drinking water, as determined by the EPA in two different surveys (EPA, 1985). Significant positive statistical correlations were found between cancer mortality specific for bladder, rectum, and large intestine and chloroform levels in treated drinking water in the "weighted" regression for white females. With white males, only stomach cancer was positively correlated with chloroform levels. The study contained no information on actual levels of chloroform present in the drinking water.

With reference to these and other epidemiological studies, EPA (1985) concluded that the association between the three cancer types and exposure to chlorinated drinking water is weak but significant. However, as exposure to chlorine in water is not the same as exposure to chloroform, the epidemiologic evidence for chloroform's carcinogenicity must be termed inadequate (EPA, 1985).

In a chronic clinical study DeSalva et al (1975) monitored subjects using dentifrice containing 3.4% chloroform and mouthwash containing 0.43% chloroform for 1 to 5 years. The estimated daily ingestion of chloroform was 0.3 to 0.96 mg/kg body weight. No hepatotoxicity was observed. In a 10- year clinical study subjects ingested 1.6 to 2.6 g of chloroform in a cough suppressant daily. This corresponded to roughly 23 to 37 mg/kg/day. Some reversible hepatotoxicity was observed (Wallace, 1959).

14.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

WHO (1984) has set a guideline value of 30 ug/litre for chloroform in drinking water. The following suggested no adverse response levels (SNARLs) have been calculated by NAS (1982). These calculations are for noncarcinogenic effects only.

24-Hour Exposure	22 mg/litre
7-Day Exposure	3.2 mg/litre
Chronic Exposure	not applicable due to carcinogenicity of chloroform.

The National Institute for Occupational Safety and Health (NIOSH) recommended in 1976 that exposure to chloroform be limited to 10 ppm as a time-weighted average for up to a 10-hour workday, 40 hour workweek. NIOSH has proposed a ceiling level of 50 ppm for any 10-minute period (EPA, 1985).

Chloroform has been approved by the FDA as an indirect food additive (EPA, 1985).

14.6. HAZARD ASSESSMENT

Levels of chloroform in Edmonton drinking water were normally below 5 ug/l but a level of 11 ug/L was reported in July of 1984. On the basis for some concern regarding the carcinogenicity of this compound, a worst case level in Edmonton water (60 kg person consuming 2 L of water containing 5 ug/L chloroform per day) would suggest an upper risk of 1.15×10^{-5} of developing cancer. Since it is generally considered that a 1×10^{-6} risk may be regarded as negligible, there is some concern that levels of chloroform in Edmonton water may represent a possible hazard. The normal levels are, however, well below the guideline value of 30 ug/L set by WHO (1984) and any risk associated with the presence of chloroform in water has been deemed negligible compared to the risk associated with inadequate disinfection (WHO, 1984). It is felt that concern regarding the presence of chloroform should be balanced against the benefits derived from the use of chlorine as a disinfectant and that, on this basis, the risk is acceptable.

15. NAME: 1,1-DICHLOROETHANE

15.1. CAS No.: 75-34-3

15.2. SOURCES AND USES

1,1-Dichloroethane is used, in relatively small quantities, as a chemical intermediate and as a solvent for extraction and degreasing (Colacci et al, 1985). NIOSH (1978) estimates that 4600 workers are exposed occupationally to 1,1-dichloroethane in the United States. 1,1-Dichloroethane does not appear to be commercially produced in the United States (NIOSH, 1978). The US EPA conducted a study on levels of volatile organic compounds in finished water supplies that use groundwater sources. Based on 280 random sample sites, 1,1-dichloroethane was detected in 3.6% of the sites at a mean value of 0.51 ug/L. The quantitation limit was 0.2 ug/L and the maximum concentration detected was 3.2 ug/L (Westrick et al, 1984). The 1,2-isomer of dichloroethane is generally considered to be of greater toxicological significance than the 1,1-isomer.

15.3. PHARMACOLOGY AND METABOLISM

The metabolic pattern of 1,1-dichloroethane has not been fully characterized. It has, however, been shown to bind to the substrate binding site of hepatic cytochrome P-450 (McCall et al., 1983). This group proposed that 1,1-dichloroethane is acted upon by the hepatic microsomal cytochrome P-450 system with acetic acid being the major metabolite formed. Minor metabolites are thought to include 2,2-dichloroethane and other mono- and dichloroacetic acids. Chloroacetaldehyde, a mutagenic species, is formed in small quantities during 1,1-dichloroethane metabolism (Colacci, 1985). Using isolated rat hepatocytes and a spin trapping technique Tomasi et al. (1984) demonstrated that free radical production was detectable under hypoxic conditions. The 1,2-isomer is able to form free radicals under normoxic conditions. 1,1-dichloroethane is not bioactivated by cytosolic GSH-transferases like some other halogenated compounds, rather conjugation with GSH is a detoxication step (Colacci et al, 1985). The 1,2-isomer is greatly bioactivated by cytosolic enzymes in the presence of GSH to reactive form(s) covalently interacting with DNA and enzymatic macromolecular constituents (Colacci et al, 1985). Mitoma et al. (1985) carried out a metabolic disposition study on 1,1-dichloroethane. Osborne-Mendel rats and B6C3F$_1$ mice were administered the maximum tolerated dose and one half the maximum tolerated dose orally 5 days per week for 4 weeks, followed by a single dose of the corresponding radiolabelled compound. In rats, 86.13 percent of the administered dose was present in the expired air, 5.12% as CO_2, and 0.92% as excreta. In addition, 1.41% was present in the carcass. In mice, 70.42% was present unaltered in the expired air, 25.23% as CO_2, 1.64% as excreta and 2.43% in the carcass. In rats, 7.45% of the administered dose was metabolised compared to 29.3% in mice. The rate of metabolism of 1,1-dichloroethane is greater than that of 1,2-dichloroethane. This is not consistent with the higher toxicity, mutagenicity and carcinogenicity of 1,2-dichloroethane (Colacci et al, 1985).

15.4. ANIMAL TOXICOLOGY

15.4.1. ACUTE

There is very limited data available in the literature on the acute toxicity of 1,1-dichloroethane. Clayton and Clayton (1981) report an oral LD_{50} of 14.1 g/kg for rats. All chloroethane compounds are known to cause central nervous system depression in laboratory animals, including unconsciousness, intoxication and irregular respiration (NIOSH, 1978). 1,1-dichloroethane has a relatively low capacity to cause liver or kidney injury (Clayton and Clayton, 1981).

15.4.2. CHRONIC

Hofmann et al. (1971) reported that rabbits, rats, guinea pigs, and cats tolerated 6-hour daily exposures to 500 ppm for 13 weeks, 5 days/week with no adverse effects. Rabbits, rats and guinea pigs tolerated an additional 13 weeks at 1000 ppm but cats showed increased blood urea and evidence of kidney injury was observed upon histological examination.

15.4.3. MUTAGENICITY AND GENOTOXICITY

Colacci et al. (1985) examined the in vivo and in vitro genotoxicity of 1,1-dichloroethane in terms of covalent binding to nucleic acids and proteins. 1,1-dichloroethane was shown to bind covalently to macromolecules of rat and mouse organs in vivo after intraperitoneal injection, with a Covalent Binding Index value in the same order as that found with weak carcinogens (Lutz, 1979). This covalent binding is catalyzed by a P-450-dependent microsomal mixed oxidase function system (McCall et al., 1983; Colacci et al., 1985). In vitro studies with rat and mouse microsomes from different organs indicate that liver microsomes, and to a lesser extent lung microsomes, mediate binding. Microsomes from kidney and stomach were ineffective. The authors (Colacci et al., 1985) concluded that further genotoxicity studies with 1,1-dichloroethane are necessary.

15.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

Schwetz et al. (1974b) examined the embryo- and fetotoxicity of inhaled 1,1-dichloroethane in female Sprague-Dawley rats. Groups of rats were exposed to either 3800 or 6000 ppm 1,1-dichloroethane 7 h/day on days 6 through 15 of gestation. No effect was seen on the incidence of fetal resorptions, fetal body measurements or on the incidence of gross or soft tissue anomalies at either dose level. A significantly increased incidence of delayed ossification of sternebrae was associated with exposure to 6000 ppm 1,1-dichloroethane. The incidence of vertebrae with bipartite centra was significantly lower in fetuses of rats exposed to 3800 ppm than in control fetuses. Both dosage groups showed statistically significant decreases in maternal food consumption and weight gain. Conception rate, number of implantations, litter size or gross appearance of the liver was not affected. The authors concluded that exposure to 1,1-dichloroethane caused little or no maternal toxicity as well as very little embryo and fetal toxicity (Schwetz et al., 1974b).

15.4.5. CARCINOGENICITY

Only one study has examined the carcinogenicity of 1,1-dichloroethane (NCI, 1978). A bioassay of technical grade 1,1-dichloroethane for possible carcinogenicity was carried out using Osborne-Mendel rats and $B6C3F_1$ mice. 1.1-dichloroethane in corn oil was administered by gavage, at either of 2 dosages, to groups of male and female animals of each species, 5 days a week for a period of 7 weeks. The high and low time-weighted average doses of 1,1-dichloroethane were as follows: 764 and 382 mg/kg body weight for male rats; 950 and 475 mg/kg body weight for female rats; 2885 and 1442 mg/kg body weight for male mice; and 3331 and 1665 mg/kg body weight for female mice. A statistically significant increase in mammary adenocarcinomas and hemangiosarcomas in female rats and hepatocellular carcinomas in mice was observed. A significant increase in the incidence of endometrial stromal polyps was observed among female mice. Survival was poor in all rat groups and several mouse groups. NCI (1978) summarized that under the conditions of this bioassay there was no conclusive evidence for the carcinogenicity of 1,1-dichloroethane to rats or mice.

IARC (1979),however, concluded that there is sufficient evidence that structurally related compound, 1,2-dichloroethane is carcinogenic in mice and rats.

15.5. HUMAN TOXICOLOGY

High doses of 1,1-dichloroethane have been reported to cause liver necrosis in humans (Colacci et al., 1985). Repeated or prolonged skin exposure to 1,1-dichloroethane can defat the skin and cause dermatitis; exposure via inhalation can result in pulmonary irritation (NIOSH, 1978). No other human data were found in the literature.

15.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

In 1980 the ACGIH recommended a TWA of 200 ppm for 1,1-dichloroethane (NIOSH, 1978). No drinking water standards were found and The National Academy of Science does not address 1,1-dichloroethane in the Drinking Water and Health documents. WHO (1984) calculated that the upper 95 percent confidence limit on a 10^{-6} lifetime risk of cancer was equivalent to 0.94 ug/L for structurally related compound, 1,2-dichloroethane.

15.6. HAZARD ASSESSMENT

Due to a lack of pivotal toxicological data it is not possible to adequately assess the human health hazards associated with 1,1-dichloroethane in Edmonton drinking water. However, it is probably appropriate to use the data on 1,2-dichloroethane for a worst case assessment. The scientific literature suggests that 1,2-dichloroethane is more hazardous than 1,1-dichloroethane and this worst case assessment is thus probably highly conservative. Levels of 1,1-dichloroethane were below the detection limits of 0.2 ug/L except for a single analysis carried out in April of 1983 when the level of 1,1-dichloroethane in Edmonton drinking water was reported to be 0.8 ug/L. This is below the concentration equivalent to a 10^{-6} lifetime cancer risk for 1,2-dichloroethane as calculated by WHO (1984). On this basis, the levels of 1,1-dichloroethane reported in Edmonton drinking water are unlikely to present a significant human health risk.

16. NAME: TRICHLOROETHYLENE (TCE)

16.1. CAS No.: 79-01-6

16.2. SOURCES AND USES

Trichloroethylene does not occur as a natural product but is commercially synthesized by the chlorination and dehydrochlorination of 1,2-dichloroethane. Trichloroethylene is used primarily in vapour degreasing of metals and as a solvent in the dry cleaning industry. Other applications include its use as a low-temperature heat transfer fluid and as a component of consumer products such as cleaning fluids and spot removers (IARC, 1979). McConnell et al. (1975) reported that trichloroethylene is widely distributed in municipal water supplies, rivers, oceans, and rainwater at levels at or less than 1 ug/l. A survey of 30 Canadian potable water treatment facilities revealed a maximum level of 9 ug/l for both raw and treated water (Otson et al., 1982).

16.3. PHARMACOLOGY AND METABOLISM

Trichloroethylene is absorbed in the body after inhalation and ingestion, and has been demonstrated to penetrate intact human skin (NAS, 1980). In rats, absorption of ingested trichloroethylene leads to urinary excretion of trichloroacetic acid (1-5%) and trichloroethanol glucuronide (10-15%), with 72-85% of the trichloroethylene expired unchanged (Daniel, 1963). During metabolism in rodents, a reactive intermediate, 2,2,2-trichloroepoxide, is formed by the action of the hepatic mixed function oxidase enzyme system. This active species can bind covalently to cytochromes as well as to various cellular macromolecules, including DNA, leading to the hepatotoxicity seen in trichloroethylene studies with rodents. Humans metabolize trichloroethylene in a manner similar to rodents. Kinetic data on trichloroethylene clearance in humans, however, suggests that humans metabolize approximately 20 times less trichloroethylene on a weight basis than rats at similar exposure levels (Stott et al., 1982). As there is a good correlation between the rate and extent of metabolism and the degree of observed hepatic toxicity, extrapolation of risk assessment to humans from rodent data must take this into consideration.

16.4. ANIMAL TOXICOLOGY

16.4.1. ACUTE

Trichloroethylene has a low acute toxicity to animals; selected values for acute toxicity assays are given below:

Chemical Form	Species	Sex	Route	Parameter	g/kg Body Wt.	References
TCE	Rat	?	Oral	LD_{50}	7.2 g/kg	IARC, 1979.
	Mouse	?	Oral	LD_{50}	2.85 g/kg	IARC, 1979

16.4.2. CHRONIC

In a variety of species of laboratory animals long-term studies indicate that repeated exposure to trichloroethylene is no more harmful than acute exposure (NAS, 1980). Stott et al. (1982) found that minimal hepatotoxic effects were evident after treatment of mice with 250 mg/kg body weight trichloroethylene by gavage 5 days/week for 3 weeks. A dose regime of 500 mg/kg or greater resulted in an increased liver weight and centrilobular hypertrophy of the liver. Rats, however, exposed to 1000 mg/kg body weight per day suffered no liver damage.

16.4.3. MUTAGENICITY AND GENOTOXICITY

The mutagenicity of trichloroethylene has been examined in various assays. Trichloroethylene was found to be non-mutagenic in the Ames Salmonella assay when tested with TA100 using vapor exposure. The assay was carried out in the presence and absence of phenobarbital-induced S9 fraction from male mice and exposure levels were as high as 20% in air (v/v) for up to 16 hours (Bartsch et al., 1979). Henschler et al. (1977) showed that two impurities of technical grade TCE, epichlorohydrin and 1,2-epoxybutane, were mutagenic in the Ames Salmonella assay in the absence of rat liver microsomes. Chloral hydrate, a trichloroethylene metabolite was found to be mutagenic in the Salmonella assay in doses ranging from 0.5 to 10.0 mg/plate (TA100). Mutagenic activity was enhanced in the presence of rat liver S9 fraction (Waskell, 1978). Negative results were obtained for chloral hydrate mutagenicity with the frameshift mutant, TA98.

Trichloroethylene induced concentration-dependent reverse mutations in Saccharomyces cerevisiae strain XV185-14C in the presence of mouse S9 fraction. The authors concluded that trichloroethylene induced both base-pair and frameshift type mutations (Shahin and Von Borstel, 1977). Using S. cerevisiae D7 and D4, Bronzetti et al. (1980) carried out host-mediated assays with mice. Both point mutations and gene conversions were observed in the D7 strain and gene conversions in the D4 strain, after recovery of metabolites from the liver and kidney following both acute and subacute dosing. S. cerevisiae recovered from lung tissue suffered little if any mutagenic effects.

A dominant lethal assay with NMRI-Han/BGA male mice exposed for 24 hours to 50, 202, and 450 ppm trichloroethylene was negative (Slacik-Erben et al., 1980)

16.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

A limited number of studies have investigated the teratogenicity of trichloroethylene to rodents. Schwetz et al. (1975) found that exposure to 300 ppm trichloroethylene in air for 7 hours per day on days 6 through 15 of gestation had no teratogenic effect on either mice or rats. In another investigation, rats exposed to 1800 ppm trichloroethylene in air for 6 hours per day on days 0 through 20 of gestation showed no signs of teratogenicity. Some evidence of fetotoxic effects, including increased incidence of displaced right ovary and incomplete ossification of the sternum, were observed (Dorfmueller et al., 1979).

16.4.5. CARCINOGENICITY

A National Cancer Institute carcinogenicity bioassay (1976a) studied the effects of trichloroethylene on male and female $B6C3F_1$ mice and Osborne-Mendel rats. Two dose levels, administered by gavage in corn oil, were investigated using 50 animals of each sex per group. The low-dosage regime for rats consisted of 650 mg/kg body weight five days a week for seven weeks, followed by 750 mg/kg body weight for nine weeks and then by 500 mg/kg body weight in a cycle of 4 weeks treatment and 1 week no treatment. The high-dosage regime was identical with double the dose level. No significant increase was noted in the occurrence of tumours at any site for the rat. In studies with mice, the male low dose group were given 1000 mg/kg body weight for 12 weeks followed by 1200 mg/kg body weight for 66 weeks. The females were given 700 mg/kg body weight for 12 weeks followed by 900 mg/kg body weight for a further 66 weeks. In the high dose groups, males and females were given double the quantities of trichloroethylene of the corresponding low dose animals. In all cases the dosing schedules were five days a week. Primary hepatocellular carcinoma were seen in significantly increased numbers in both treated male and female mice. The incidence in males was 64.5% (31/48) at the high dose and 52% (26/50) at the low dose, compared to control values of 5% (1/20) and 6.5% (5/77) respectively. The corresponding values for females were 23% (11/47) at the high dose level and 8% (4/50) at the low dose, as compared to 1/80 for the colony controls. Metastases of the liver tumours was seen in 14% (7/98) of the treated males. An increase in the number of primary tumours of the lung was seen in treated animals (predominately benign adenoma) though this increase was not statistically significant.

Several concerns regarding the design of the NCI (1976a) carcinogenesis bioassay of trichloroethylene have been raised. Henschler et al. (1977) found that the industrial grade trichloroethylene used in the NCI study contained approximately 0.2% of both epichlorohydrin and 1,2-epoxybutane, both potent mutagens in the S. typhimurium mutagenicity assay. As well, lesser amounts of chloroform and carbon tetrachloride were found to be present. Other concerns included the daily administration of such extremely large quantities of a chemical and the questionable practice of housing subjects in the same room as animals receiving a variety of other volatile chemicals. IARC (1979) concluded that there is limited evidence for the carcinogenicity of trichloroethylene to mice.

A subsequent trichloroethylene carcinogenicity bioassay (NTP, 1982a) included the B6C3F$_1$ mouse and the Osborne-Mendel rat in an attempt to clarify ambiguities in the NCI (1976a) report. In these studies a single 1000 mg/kg body weight dose of trichloroethylene in corn oil was administered by gavage to 50 mice of each sex, and doses of 1000 and 500 mg/kg body weight were given in the same manner to 50 rats of each sex, 5 days/week for two years. Each group of animals was housed in a separate room and no detectable traces of 1,2-epoxybutane or epichlorohydrin was present in the trichloroethylene. The results observed in the mice support the previous NCI (1976a) finding that trichloroethylene significantly increased the incidence of hepatocellular carcinomas in B6C3F$_1$ mice of both sexes. It appeared that the other subjects tested, namely Marshall, ACI, Osborne-Mendel, and Fisher 344 rats have a much lower sensitivity to the carcinogenicity of trichloroethylene than the B6C3F$_1$ mouse. Stott et al. (1982) suggest this difference may be due to the increased ability of the B6C3F$_1$ strain to metabolize trichloroethylene to a reactive compound.

Henschler et al. (1980) carried out inhalation carcinogenicity bioassays with 3 animals: rat, mouse and Syrian hamster. Two dose levels were investigated, 100 ppm and 500 ppm (6 hours/day, 5 days/week) for 18 months with 30 animals of each sex and species used at each dose level, together with a similarly sized control group. No significant increase in the incidence of tumours of any type was noted in the rat or Syrian hamster. A significant increase in the incidence of lymphomas was observed in female mice, with 64% (18/28) found at 500 ppm and 57% (17/30) found at 100 ppm, as compared to 31% (9/29) in the controls. These results suggest that trichloroethylene produced an increase in the incidence of lymphomas in female mice; however this type of tumour occurs spontaneously in female mice of this strain with a high frequency. The value of this study is severely limited due to the small sample size and the low two-year survival rate, especially for females.

On the basis of the NTP study (1982a), the National Academy of Science (1983) calculated a lifetime carcinogenic risk estimate of 5.48 x 10^{-7} (95%

upper confidence limit). This value is based on daily consumption of 1 L of water containing 1 ug/L by a 70 kg man and is compared to a risk of 1.0 x 10^{-6}, which is considered negligible. Daily consumption of 2 L of water containing 1 ug/L trichloroethylene would correlate with a lifetime carcinogenic risk estimate of 1.096 x 10^{-6}.

16.5. HUMAN TOXICOLOGY

IARC (1979) summarized the results of a Swedish epidemiological study of cancer mortality among 518 males exposed occupationally to relatively low levels of trichloroethylene. The mortality data of the experimental cohort was compared with mortality data from national statistics. This study provided no indication of any increased risk of death from cancer or any other causes in the exposed group. A similar study in Finland (Tola et al., 1980) with a sample size of 2205 persons failed to show any relationship between trichloroethylene exposure and cancer. Severe limitations in these studies, including concomitant exposure to other chemicals, prevents any conclusions being drawn regarding the carcinogenicity of TCE.

Several cases of human ingestion of trichloroethylene have been documented in the literature. Clinical symptoms involve gastrointestinal upset, narcosis, and occasional cardiac abnormalities (NAS, 1980). Inhalation experiments have exposed human subjects to up to 500 ppm trichloroethylene vapour. Impairment of psychophysiological functions and mucus membrane irritation were observed (NAS, 1980).

16.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

NAS (1980) has suggested the following suggested no-adverse response levels (SNARLs) for drinking water:

24 h exposure	105 mg/L
7 d exposure	15 mg/L
Chronic exposure	insufficient data available

16.6. HAZARD ASSESSMENT

Pivotal to the carcinogenic risk assessment of this chemical is the observation that rates of metabolism are lower in man than in laboratory animals. This would result in lower rates of conversion into toxic metabolites which may reduce the carcinogenic risk below that calculated by NAS (1983). Levels of trichloroethylene in Edmonton water were all reported to be less than the detection threshold of 0.2 ug/L (ppb) except for one report of 0.4 ug/L in March 1984. On the basis for some concern regarding the carcinogenicity of this compound, and a worst case continuous level of 0.4 ug/L in Edmonton water (60 kg person consuming 2 L/day) would suggest an upper 95% confidence limit of the risk of 4.4×10^{-7} of developing cancer. Since it is generally considered that 1×10^{-6} risk may be regarded as negligible, it is unlikely that levels of trichloroethylene in Edmonton water may represent a human health risk.

17. NAME: TETRACHLOROETHYLENE

17.1. CAS No.: 127-18-4

17.2. SOURCES AND USES

This chemical is widely used in industry as a solvent (particularly in the dry cleaning industry), as a heat exchanger and as a feedstock for the synthesis of other chemicals such as fluorocarbons (NAS, 1979). It may enter water as a contaminant or may be formed during the chlorination process. It has been reported in finished water from US cities at levels from 0.07 to 5 ug/L (NAS, 1977).

17.3. PHARMACOLOGY AND METABOLISM

Tetrachloroethylene is readily absorbed through the lungs by humans (Fernandez et al., 1976; Ohtsuki et al., 1983; Stewart et al., 1961), dogs (Hobara et al., 1983), and rodents (Pegg et al., 1979; Schumann et al., 1980), through the skin by humans (Stewart and Dodd, 1964) and after oral administration to rodents (Frantz and Watanabe, 1983; Pegg et al., 1979; Schumann et al., 1980).

The major route of elimination of tetrachloroethylene is the expiration of unchanged tetrachloroethylene. For example, after single oral or inhalation exposures to tetrachloroethylene 70 to 98% of the body burden was eliminated in the expired air (Pegg et al., 1979; Daniel, 1963).

Studies have been conducted in rats and mice in an attempt to explain the sensitivity of mice and the resistance of rats to the development of hepatic tumors after exposure to tetrachloroethylene. Mice were found to metabolize 8.5 and 1.6 times more tetrachloroethylene per kilogram of body weight after inhalation of 10 ppm tetrachloroethylene or a single oral dose of 500 mg/kg tetrachloroethylene, respectively (Schumann et al., 1980). The more extensive metabolism of tetrachloroethylene in mice than in rats is associated with a greater susceptibility of mice than rats to liver damage caused by tetrachloroethylene and with increased hepatic DNA synthesis in mice compared to rats after treatment with tetrachloroethylene. Similarly, after oral treatment of rats or mice with tetrachloroethylene for five times a week for four weeks, mice metabolized more tetrachloroethylene than rats (22.25% vs. 5.1%) and eliminated less tetrachloroethylene in the expired air (57.55% vs 79.25%).

Studies on the metabolism of tetrachloroethylene have identified a number of metabolites such as trichloroacetic acid, oxalic acid, and dichloroethylene (trace, Yllner, 1961) as well as inorganic chloride (Daniel, 1963). Daniel (1963) proposed that tetrachloroethylene was metabolized by expoxidation to tetrachloroethylene oxide. He suggested that subsequent metabolism would yield trichloroacetyl chloride which may be hydrolyzed to yield trichloroacetic acid and inorganic chloride. Dekant _et_ _al_. (1986) have recently reexamined the metabolism of tetrachloroethylene in rats and mice using more sophisticated techniques. In these experiments, the animals were given a single oral dose (800 mg/kg body weight) of [^{14}C]-tetrachloroethylene in corn oil, and the elimination and metabolism of tetrachloroethylene studied by HPLC and GC/MS. In confirmation of earlier studies, the expiration of unchanged tetrachloroethylene was the major route of elimination and amounted to 91.2 and 85.1 percent of the dose in rats and mice, respectively. Only trace amounts of carbon dioxide were found. Total urinary metabolites amounted to 2.3 and 7.1 percent of the dose in rats and mice, respectively. These metabolites were identified as oxalic acid (8.0% of urinary radioactivity in rats; 2.9% in mice), dichloroacetic acid (5.1% in rats; 4.4% in mice), trichloroacetic acid (54% in rats; 57.8% in mice), _N_-trichloroacetyl-aminoethanol (5.4% in rats; 5.7% in mice), free and conjugated trichloroethanol (8.75 in rats; 8.0% in mice), _N_-acetyl-_S_-(1,2,2-trichlorovinyl-L-cysteine (1.6% in rats; 0.5% in mice), and an unidentified conjugate of trichloroacetic acid.

As in rodents, trichloroacetic acid and trichloroethanol have been identified as the major urinary metabolites of tetrachloroethylene in man (Fernandez _et_ _al_., 1976; Ikeda and Ohtsuji, 1972; Ohtsuki _et_ _al_., 1983). The urinary biological half-life of tetrachloroethylene in man is estimated to be 144 hr (Ikeda, 1977).

There is a general relationship between the extent of metabolism of tetrachloroethylene and its hepatotoxicity (Buben and O'Flaherty, 1985). The covalent binding of $[^{14}C]$-tetrachloroethylene metabolites to tissue proteins has been observed in in vivo studies (Schumann et al., 1980) and in in vitro studies with the isolated, perfused rat liver (Bonse et al., 1975) and with hepatic microsomal fractions (Bolt and Link, 1980; Costa and Ivanetich, 1980). This covalent binding is presumably due to the acylation of tissue nucleophiles by trichloroacetyl chloride, because hydrolysis of tissue fractions yielded trichloroacetic acid. The covalent binding of tetrachloroethylene-derived metabolites to DNA has been sought, but not observed (Schumann et al., 1980).

17.4. ANIMAL TOXICOLOGY

17.4.1. ACUTE

This compound has relatively low acute toxicity in mammals and representative toxicity data is given below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Tetra-chloro-ethylene	dogs	?	Oral	LD_{50}	4000	NAS, 1977
	rabbits	?	Oral	LD_{50}	5000	NAS, 1977
	humans	M/F	Oral	Intoxication	70	NAS, 1980

17.4.2. CHRONIC

Oral dosing with tetrachloroethylene at levels of 380 to 1070 mg/kg body weight in rats and mice for periods of up to 78 weeks resulted in a high incidence of nephrotoxicity but little evidence of hepatotoxicity (NCI, 1977). Doses of 100 to 1000 mg/kg body weight tetrachloroethylene given daily over 11 days to $B6C3F_1$ mice and SD rats caused increases in liver weight at all doses above 100 mg/kg body weight. Hepatic lesions were observed in the mice at all doses but were only apparent in rats receiving 1000 mg/kg body weight/day (Schumann et al., 1980).

17.4.3. MUTAGENICITY AND GENOTOXICITY

The genotoxicity of tetrachloroethylene has been studied extensively in both in vitro and in vivo test systems. The most thorough and up to date testing in in vitro tests are the studies of Shimada et al., (1985) using several of the Ames tester strains. In these studies stabilized tetrachloroethylene (99.80-99.93% pure) produce a marginally (3-10 fold) positive response in TA1535 and TA100 (base-pair substitution) at cytotoxic doses. These results did not demonstrate a dose-response relationship and the response did not occur below the cytotoxic dose of 2.5% (v/v). Higher purity stabilized tetrachloroethylene (99.98 +% pure) did not produce any genotoxic response. These observations suggest that the stabilizers may have contributed to the weak genotoxic response observed with tetrachloroethylene of lower purity. This conclusion is supported by earlier observations of Margard, (1978) who also found that stabilized tetrachloroethylene produced a weak response at cytotoxic doses in tester strains TA100, TA1538 and TA98 whereas highly purified (no detectable impurities) tetrachloroethylene did not. The impurities/stabilizers in the lower grade tetrachloroethylene tested by Margard (1978) contained, in addition to some of those found in the Shimada et al. (1985) study, 0.07% epichlorohydrin and 0.007% N-methylmorpholine (EPA, 1984). Thus it appears that stabilizers in commercial tetrachloroethylene may be responsible, in whole, or in part for its weak mutagenicity in the Ames system. Tests in Salmonella typhimurium have been uniformly negative (EPA, 1984). A host-mediated assay using a Salmonella tester strain was reported to produce positive results but the results were not consistent between male and female mice and no dose response was observed (Beliles et al., 1980).

Studies of reverse mutation in Saccharomyces cerevisiae have produced both positive and negative results with the positive results occurring predominantly at toxic doses. Overall there is no strong evidence that tetrachloroethylene is genotoxic in yeast.

Assays for DNA damage and repair using both rat hepatocytes and human lymphocytes have produced variable results. Tetrachloroethylene appeared to produce positive results for unscheduled DNA synthesis in rat hepatocytes in culture when administered at high concentration (close to cytotoxic doses) in the vapour phase but not in liquid phase exposure, a result that is difficult to rationalize (Shimada *et al.*,1985). Other studies of unscheduled DNA synthesis in rat hepatocytes have produced consistently negative results (Costa and Ivanetich, 1984). Perocco *et al.* (1985) noted, with human lymphocytes in culture, that tetrachloroethylene induced toxicity and reduced thymidine uptake in the absence of S-9 microsomal fraction at levels ranging from 10^{-2} - 10^{-4} molar but to a lesser extent in the presence of a metabolic activating system. The significance of these findings in relation to the genotoxicity of tetrachloroethylene is unclear. *In vivo* studies of tetrachloroethylene genotoxicity have produced negative results in *Drosophila* and in a rat dominant lethal assay.

No convincing evidence that tetrachloroethylene is genotoxic *in vitro* or *in vivo* has been found in the literature. Positive results when obtained have occurred only at cytotoxic doses and are confounded by the presence of potentially mutagenic impurities/stabilizers in commercial tetrachloroethylene. The negative results of genotoxicity testing of commercial tetrachloroethylene *in vivo* indicate that, at the concentrations present, the impurities/stabilizers produced no detectable response. Overall the risk of tetrachloroethylene genotoxicity is considered to be negligible.

Studies on the genotoxicity of tetrachloroethylene metabolites have been carried out. Tetrachloroethylene-oxide was found to produce a dose-dependent increase in a number of revertants in *Salmonella* TA 1535 at concentrations ranging from 2.5-5.0 mM. Testing of trichloroacetic acid, the final end product of metabolism has produced negative results in a *Salmonella* assay (Waskell, 1978). Tetrachloroethylene-oxide was found to produce positive effects in a *E. coli* pol A assay but did not increase the number of revertants in *E. coli* WP2 vvr A (Kline *et al.*, 1982). A recently identified metabolite of tetrachloroethylene, *S*-(1,2,2-trichlorovinyl)-L-cysteine (Dekant *et al.*, 1986) appears to be mutagenic to *S. typhimurium* TA100 in both the presence and absence of rat S9 kidney microsomal fractions (Odum and Green, 1985).

17.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No oral exposure studies regarding the teratogenicity of tetrachloroethylene were found in the literature. However, exposure of mice and rats to concentrations of 300 ppm in air (7 h/day from days 6 to 15 of gestation) failed to indicate any teratogenicity (Schwetz et al., 1975). Exposure of rats to concentrations of 100 to 900 ppm tetrachloroethylene in air for 7 h/day from day 14 to 20 of gestation demonstrated some behavioral effects in the pups born to the rats exposed at the high concentration but not at the low concentration (Nelson et al., 1979). This was associated with lower rates of weight gain in the mothers and may have been mediated through maternal toxicity or anorexia. NAS (1983) concluded that the limited data indicate that tetrachloroethylene is not teratogenic in rats or mice.

17.4.5. CARCINOGENICITY

Tetrachloroethylene has not been reported to induce tumors in laboratory animals via the inhalation route (NAS, 1980), however, liver tumors were reported in a study which used oral dosing (NCI, 1977). Rats and mice were given doses from 386-1072 mg/kg body weight/day by gavage for up to 78 weeks. A high, dose-dependent incidence of hepatocellular carcinoma was reported in the mice but not the rats. The doses of tetrachloroethylene used were close to the toxic level as evidenced by a dose-dependent increase in mortality in the experimental animals.

17.5. HUMAN TOXICOLOGY

Several epidemiological studies on tetrachloroethylene have been carried out. Katz and Jowett (1981) reported a study of female laundry and dry cleaning workers in Wisconsin which was designed to assess retrospective proportional mortality. The study indicated that kidney malignancy was elevated at $P = 0.05$ in a subset of the exposed group, however,the use of multiple testing and, more seriously, the lack of accurate exposure or length of exposure data for tetrachloroethylene also weaken the conclusions of this study. Blair *et al.*, (1979) reported a proportional mortality study based on the causes of death among laundry and dry cleaning workers. Results of this study indicated a possible increased risk of cancer of the cervix, lungs and skin. However, the results were not statistically significant because of multiple testing. Brown and Kaplan (1985) conducted a study based on a retrospective cohort analysis conducted in dry cleaners who had at least one year exposure to tetrachloroethylene before 1960. Results suggested a somewhat lower (compared to the general population) death rate from cancer and the incidence of cancer of liver was less than expected. However, there was some evidence for increases in all cancers and particularly cancer of the urinary and genital system with increasing duration of exposure and time since first exposure.

Most of the epidemiological studies on tetrachloroethylene were characterized by relatively small numbers and very poor information about previous exposure which, in most instances, was mixed. For this reason it is difficult to draw inferences about the specific effects of tetrachloroethylene exposure. The reported positive findings are more than likely to be the result of multiple testing and these studies should be viewed as hypothesis generating, rather than as answering specific questions. There is thus no positive evidence in human studies that tetrachloroethylene has been shown to cause increased rates of cancer. Equally, the data are unable to provide adequate evidence against an increase in cancer rates.

17.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

NAS (1980) suggested the following no adverse effect levels (SNARLS) for tetrachloroethylene in water:

24 h exposure	172 mg/L	NAS, 1980
7 d exposure	24.5	NAS, 1980

NAS (1983) was not able to set a chronic SNARL on the basis of lack of sufficient evidence

17.6. HAZARD ASSESSMENT

On the basis of the NCI (1977) cancer study in mice, NAS (1979) calculated the risks of developing cancer from a lifetime of exposure to tetrachloroethylene in humans. The mouse data was corrected for dose regimen and for body weight/surface area. They reported an upper 95% confidence estimate of the lifetime risk of cancer from consumption of water containing 1 ug/L of 1.4×10^{-7}. The concentration of tetrachloroethylene in Edmonton drinking water was consistently reported to be less than the detection threshold of 0.2 ppb (0.2 ug/L). On the assumption of worst case this would be equivalent to an upper 95% confidence estimate of lifetime cancer risk of 2.8×10^{-8} which well below the 1×10^{-6} risk generally regarded as negligible. It should also be pointed out that these calculations are based on limited evidence. Carcinogenicity has only been reported in the mouse and this has been suggested to be due to the higher rates of metabolism of tetrachloroethylene in this animal. It has not been shown that human metabolic pathways and rates of metabolism are similar to those in mice. Tetrachloroethylene is widely used in the dry cleaning industry and the widespread occupational exposure has allowed some epidemiological studies to be carried out. On the basis of the greater levels of exposure in this industry, compared to those in water and the lack of clear evidence of toxic effects, it seems very unlikely that the present concentrations of tetrachloroethylene in Edmonton drinking water are a significant health hazard to humans.

18. NAME: HEXADECANOIC ACID

18.1. CAS No.: 57-10-3

18.2. SOURCES AND USES

Hexadecanoic acid (palmitic acid) is the most abundant saturated fatty acid found in nature. Fatty acids are an essential component of saponifiable lipids found in animals, plants and microorganisms. Hexadecanoic acid can be obtained from palm oil, Japan wax, or Chinese vegetable tallow and is used in the manufacture of soaps and lubricating oils. As well, hexadecanoic acid is used in the synthesis of metal palmitates and food-grade additives (Clayton and Clayton, 1981).

18.3. PHARMACOLOGY AND METABOLISM

Hexadecanoic acid is rapidly metabolized, primarily through Beta-oxidation. Interconversion to stearic, oleic, palmitoleic, and myristic acids occurs in the liver and intestinal mucosa. After oxidation or conversion to other long chain fatty acids the carbon skeleton of hexadecanoic acid is esterified or returned to the plasma, depending on the nutritional state of the organism. The products of esterification are primarily neutral fats (Bjorkhem *et al.*, 1978).

18.4. ANIMAL TOXICOLOGY

18.4.1. ACUTE

Little information is available on the acute toxic effects of palmitic acid. The LD_{50} for intraveinous administration to mice is 57±3.4 mg/kg body weight (Windholz, 1983).

18.4.2. CHRONIC

Depression of growth was observed when weanling mice were fed diets containing 5 to 40 percent hexadecanoic acid. High mortality resulted at dietary levels of 20 to 40 percent, and 100 percent mortality was observed at the 40 percent level (Tove, 1964).

18.4.3. MUTAGENICITY AND GENOTOXICITY

No information was found in the literature on the mutagenicity and genotoxicity of hexadecanoic acid.

18.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No information was found in the literature on the teratogenicity and reproductive toxicology of hexadecanoic acid.

18.4.5. CARCINOGENICITY

Studies on the carcinogenic potential of hexadecanoic acid were negative (Clayton and Clayton, 1981).

18.5. HUMAN TOXICOLOGY

Application of 75 mg hexadecanoic acid to human skin over 3 days resulted in mild irritation (Drill and Lazar, 1977).

18.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

Hexadecanoic acid is a naturally occurring fatty acid component of animal fats and vegetable oils and has been granted GRAS status (generally recognized as safe) by the FDA for use as a direct food additive (Clayton and Clayton, 1981).

18.6. HAZARD ASSESSMENT

The paucity of toxicological data does not allow a full health hazard assessment, however, the substance is generally recognized as safe and occurs naturally in the body and a number of foods. Although levels present in Edmonton drinking water were normally below the level of detection (1.0 ppb or 1 ug/L), one instance of levels less than 10 ug/L but greater than 1 ug/L was reported. These concentrations do not suggest a human health hazard.

19. NAME: CYCLOHEXANECARBOXYLIC ACID

19.1. CAS No.: 98-89-5

19.2. SOURCES AND USES

Cyclohexanecarboxylic acid is used industrially as a solubilizer for vulcanized rubber, as a clarifier for mineral oils and as a component of insecticide formulation (Windholz, 1983). Cyclohexanecarboxylic acid has not been reported from water supplies and has not been reviewed by NAS (NAS, 1977, 1980, 1982, 1983).

19.3. PHARMACOLOGY AND METABOLISM

Little information on cyclohexanecarboxylic acid was found in the literature, however, the chemical is of toxicological interest as a metabolite of shikimic acid, a constituent of many dietary fruits and plants and which has been shown to produce stomach and hemopoietic cancers in mice (Evans and Osman, 1974). Brewster _et_ _al_. (1977a) have shown that cyclohexanecarboxylic acid is metabolized in isolated perfused rat livers into a number of metabolites such as hippuric acid and benzoic acid. This indicates that animals have the capacity for aromatization of this substance. Metabolism and dissipation was rapid and followed two major pathways, that at lower concentrations leading to the formation of hippuric acid as an endpoint. This has also been observed in _in_ _vivo_ studies in rats (Brewster _et_ _al_., 1977b).

19.4. ANIMAL TOXICOLOGY

19.4.1. ACUTE

No data were available.

19.4.2. CHRONIC

No data were available.

19.4.3. MUTAGENICITY AND GENOTOXICITY

No data were available.

19.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No data were available.

19.4.5. CARCINOGENICITY

No data were available.

19.5. HUMAN TOXICOLOGY

No data were available.

19.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No data were available.

19.6. HAZARD ASSESSMENT

There is insufficient data available for a complete human health assessment on cyclohexanecarboxylic acid. Levels reported from Edmonton drinking water were less than the detection threshold of 15 ppb (15 ug/L). This would indicate a worst case consumption of 0.5 ug/kg body weight/day in a 60 kg person consuming 2 L of water a day. Given the very limited data available, the fact that cyclohexanecarboxylic acid is rapidly metabolised by mammalian liver and is a normal metabolite of a substance which occurs in food plants for humans, it probably does not represent a significant health hazard in Edmonton drinking water. Concern is, however, expressed at the lack of an adequate toxicology data base.

20. NAME: PHTHALATE ESTERS

A number of chemically different phthalate esters are utilized in the plastics and related industries. The data on di(2-ethylhexyl) phthalate, is the most complete and the major focus of this toxicity profile will thus be on this chemical. Data on dibutyl phthalate will be discussed although less is available.

20.1.	CAS No.:	di(2-ethylhexyl) phthalate (DEHP)	117-81-7
		di-n-butyl phthalate (DBP)	84-74-2

20.2. SOURCES AND USES

DEHP and the other phthalate esters are used as plasticizers in the preparation of polyvinylchloride and other plastics to give the finished plastic product flexibility. DEHP can comprise up to 50% by weight of commercial polyvinylchloride products (Kluwe et al., 1982a). Plasticizers are not chemically bound in the polymer but are dispersed in the matrix to decrease the physico-chemical interaction of adjacent chains of the polymer. As a result, plasticizers can migrate from the polymer matrix and become dispersed in the environment. Phthalates are used in the cosmetic industry as perfume solvents and fixatives, as a suspension agent for solids in aerosols, as an antifoaming agent and as a lubricant for aerosol valves. In 1981, DBP was reported as an ingredient in a total of 590 cosmetic formulations at concentrations ranging from less than 0.1 % to between 10 and 25% (FDA, 1981). IARC (1982) reports that phthalates are also used as dielectrics in capacitors, as a pesticide carrier and in respirator fit kits. Phthalates are ubiquitous in the environment and human exposure is likely. They may also be formed as normal metabolites in certain plants and organisms.

20.3. PHARMACOLOGY AND METABOLISM

Phthalate esters are readily absorbed following oral administration. Esterase enzymes in the gastrointestinal tract are important in the absorption of phthalates. The majority of the available data indicate that the hydrolysis products, rather than the parent compound, are the entities actually absorbed into the body (Kluwe et al., 1982a: Albro et al., 1983: Huff and Kluwe, 1984). No absorption threshold for DEHP was detected in mice with doses up to 1000 mg DEHP/kg body weight (Albro et al., 1982c). Monoesteric forms were more readily absorbed than diesteric forms. The toxicity of di-n-butyl phthalate was not appreciably affected by inhibition of intestinal esterases, indicating that hydrolysis of this particular phthalate is not essential prior to absorption. In these studies, greater than 95% of the circulating phthalate was the mono-n-butyl ester, indicating that hydrolysis occurred rapidly in other body tissues (White et al., 1983). The small amounts of diesteric phthalates absorbed from the gastrointestinal tract were bound to lipoproteins in the blood whereas the monoesteric forms were either free in the serum or weakly bound to albumin. Diesteric phthalates were rapidly hydrolyzed to monoesters. Small doses (about 3 mg) of DEHP were rapidly cleared from plasma in monkeys and 72% to 88% of the dose was excreted in urine within 24 hours. The major urinary metabolites identified were the isohexanol mono-ester and MEHP and 80% of the total urinary metabolites were excreted as glucuronide conjugates. Similar data were obtained in human subjects exposed to DEHP except that the urine contained more fully oxidized metabolites (Peck and Albro, 1982).

Metabolism of DEHP to the mono-ester (MEHP) has been demonstrated in stored human blood (Rock et al., 1978: Peck et al., 1979). Hydrolysis of DEHP to 2-ethylhexanol and MEHP has been reported in the gut, liver and kidneys of the rat, ferret, baboon and humans (Albro and Thomas, 1973: Rowland, 1974: Carter et al., 1974: Lake et al., 1977: Rowland et al., 1977: Thomas and Northup, 1982: Gollamudi et al., 1983: Huff and Kluwe, 1984).

Lhuguenot et al. (1985) have recently demonstrated the metabolism of DEHP into mono-(2-ethyl-5-oxohexyl) phthalate and mono-(2-ethyl-5-hydroxyhexyl) phthalate in the rat. These metabolites were found to cause a 7- to 11-fold induction of peroxisomal activity in a primary rat hepatocyte culture system. Should this induction also occur in human liver cells, the rate and degree to which these metabolites are produced could act as an indicator of carcinogenic risk in humans.

Tanaka et al. (1978) studied the metabolism of di-n-butyl phthalate in male rats. Labeled DBP (^{14}C) was administered orally in dimethyl sulfoxide in a dose of 60 mg/kg or intravenously in saline in a dose of 10 mg/kg body weight. Of the administered radioactivity, 81.4 to 97.7% was excreted in the urine and 1.0 to 8.2% in the feces in the first 24 h after either oral or intravenous administration. No organ had any significant affinity for accumulation.

It was concluded that the hydrolysis of DBP to MBP occurred in the liver, that there was enterohepatic circulation of DBP and its metabolites and good absorption from the intestine, and that MBP was the main metabolite of DBP and was primarily excreted in urine (Tanaka et al, 1978). Albro and Moore (1974) administered DBP by gavage to male rats in two doses of 0.2 ml 24 hours apart. DBP and its metabolites were quantitated 48 hours after the first dose and, at this time 24.6% of the phthalate moiety was recovered in the urine. This consisted of 89.8% monobutyl phthalate (MBP), 2.7% phthalic acid (PA), 0.4% DBP and four other metabolites in very small quantities. The authors suggested that DBP was metabolized by hydrolysis of one ester bond and both terminal and subterminal oxidation of the remaining alkyl chain resulted in primary and secondary alcohols which presumably were further oxidized to acids and ketones.

Elder (1985) noted that there was a good correlation between rate of phthalate hydrolysis and the acute oral toxicity to rats that is reported in the literature. The more rapidly hydrolyzed phthalate esters were more toxic.

20.4. TOXICOLOGY

20.4.1. ACUTE

Phthalate esters showed a very low acute toxicity as indicated by their LD_{50} values. Phthalate esters with longer and branch chain alcohols were generally less toxic. Acute toxicity indices from selected references are indicated below.

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
DBP	mouse		IP	LD$_{50}$	3300	Autian, 1982
			IP		3800	Autian, 1982
			oral	LD$_{50}$	9000	Elder, 1985
	rat		oral	LD$_{50}$	14950	Elder, 1985
DEHP	mouse		oral	LD$_{50}$	30000	NIOSH, 1982
			IP		4200	Autian, 1973
					37500	Autian, 1982
			IV		1060	NIOSH, 1982
	rat		oral		1000	NIOSH, 1982
			IP		30700	NIOSH, 1982
					50[a]	Autian, 1973
			IV		250	NIOSH, 1982
	guinea pig		oral		26000	NIOSH, 1982
			skin		10000	Autian, 1973
	rabbit		oral		34000	Autian, 1973
			skin		25000	NIOSH, 1982

a = mL/kg

20.4.2. CHRONIC

Toxicity expressed as a chronic LD$_{50}$ did not vary to nearly the extent among various phthalate esters as did the acute LD$_{50}$. Chronic LD$_{50}$s were determined in mice by administering the compound intraperitoneally, five days/week and calculating an apparent LD$_{50}$ at the end of each week. The dose associated with a constant apparent LD$_{50}$ over 3 consecutive weeks following a minimum of 10 weeks of treatment was considered an estimate of the chronic LD$_{50}$ of the particular phthalate ester. A cumulative toxicity factor was then calculated from the ratio of the acute/chronic LD$_{50}$ values (Autian, 1982) which indicated that the cumulative toxicity factor for short-chain phthalate esters (e. g. di-n-butyl, diisobutyl-) ranged from 2.05 to 4.01. However, the cumulative toxicity factors di(2-ethylhexyl) phthalate was 27.99 mg/kg, a substantial increase in the toxicity to mice under chronic exposure conditions (Autian, 1982).

Chronic toxicity studies have been reported with DEHP in the rat (2 years exposure), guinea pig (1 year exposure) and dog (1 year exposure). No observed adverse effect levels of exposure were 60 to 200 mg/kg body weight-/day, 600 mg/kg body weight and 600 mg/kg body weight, respectively in the rat, guinea pig and dog (Carpenter et al., 1953). Decreased body weight gains were reported in male rats that received 322 or 674 mg DEHP/kg body weight/day and in females that received 774 mg DEHP/kg body weight/day after about 20 weeks exposure. Food consumption was slightly decreased in both male and female rats in these studies (Kluwe et al., 1982a).

High dosages of DEHP (1000 - 2000 mg/kg body weight/day) to rodents caused testicular and hepatic damage, including inhibition of cholesterol, phospholipid and triglyceride synthesis (Oishi and Hirage, 1980a: Mangham et al., 1981: Bell, 1982), hepatomegaly and peroxisome proliferation (Autian, 1973: Nikonorow et al., 1973: Mangham et al., 1981). Di-n-butyl phthalate (Nikonorow et al., 1973) had similar effects. Gangolli (1982) found that DBP produced testicular atrophy in the rat, mouse, guinea pig, and ferret, but not in the hamster after oral administration in a dose of 2.0 g/kg body weight per day for 10 days. Studies by Cater et al. (1977) on DBP showed that the oral administration of DBP at daily dose levels of 500 and 1000 mg/kg body weight resulted in a significant reduction in the relative testes weight within 6 days and 4 days, respectively.

The no observable effect level (NOEL) for chronic exposure in rats, guinea pigs and dogs was 60-200, 600 and 600 mg/kg body weight, respectively. The major effects of chronic exposure were decreased body weight gains and hepatomegaly with peroxisome proliferation in rodents. The early phases of hepatomegaly were hyperplastic and later phases hypertrophic in nature. Species differences have been observed in hepatic effects of DEHP. Liver enlargement was observed in both rats and hamsters treated with 25 - 1000 mg DEHP/kg body weight/day for 14 days however, the response was greater in rats than hamsters. Increased peroxisomal enzyme activities (palmitoyl-CoA oxidase and carnitine acetyltransferase) were reported in rats exposed to 100 and 250 mg DEHP/kg body weight, whereas a much less pronounced effect on these enzymes was evident in the hamster after treatment with 1000 mg DEHP/kg body weight. Histopathological assessment indicated a proportionately greater proliferation of peroxisomes in the rat than the hamster. Rat hepatic and small intestinal mucosal preparations hydrolyzed DEHP more rapidly than similar preparations from hamsters. Induction of enzymes involved in fatty acid oxidation, catalase, hepatomegaly and peroxisome proliferation were not observed in marmosets treated with DEHP (Holloway et al., 1982a & 1982b). No effects were observed on oxidative enzymes (palmitoyl-CoA-dehydrogenase) or total catalase activity in marmosets exposed to 2000 mg DEHP/kg body weight/day for 14 days (Nixon et al., 1982a and 1982b). The difference in sensitivity to the toxic effects of DEHP between marmosets and rodents may have been due to lower bioavailability of DEHP in marmosets or differences in their physiological and pharmacological response to the chemical (Jackh et al., 1984). Studies where the DEHP was administered to marmosets intraperitoneally confirmed the difference in toxicological response compared to rats, irrespective of variations in bioavailability (Nixon et al., 1982b). No data were available on the effects of phthalates on peroxisomes in human liver. Other compounds that stimulate peroxisome proliferation in rodents, however, do not produce such effects in humans.

Biochemical effects in rodents include inhibition of benzo[a]pyrene and n-hexane metabolism with a corresponding decrease in the levels of associated cytochrome P-450 isozymes. Fatty acid omega and omega-1 oxidation in the liver is increased with a corresponding increase in the levels of the associated cytochrome P-450 isozymes.

Lawrence et al. (1975) injected a series of doses of DBP intraperitoneally to groups of 10 male mice 5 days a week. The apparent LD_{50} was calculated each week until it remained constant for 3 weeks; this was the chronic LD_{50}. A chronic LD_{50} of 0.85 ml/kg body weight per day 5 days a week was reached after 25 weeks.

20.4.3. MUTAGENICITY AND GENOTOXICITY

Several tests for mutagenicity in DEHP, monoethylhexyl phthalate (MEHP) and 2-ethylhexanol conducted with *Salmonella typhimurium* (using TA1535, TA1537, TA1538, TA98, or TA100) yielded consistently negative results for mutagenicity (McCann *et al.*, 1975: Simmon *et al.*, 1977: Ruddick *et al.*, 1981: Kozumbo *et al.*, 1982: Zeiger *et al.*, 1982: Seed, 1982: Hopkins, 1983: Kirby *et al.*, 1983: Yoshikawa *et al.*, 1983). Contrary to these findings, Tomita *et al.* (1982) reported that MEHP was mutagenic in *Salmonella typhimurium*. Positive mutagenic results were obtained when the mutagenesis test was conducted with DEHP and the S-9 microsomal fraction from induced rat liver which metabolized DEHP to MEHP. The same S-9 preparation also eliminated the mutagenic activity of MEHP, presumably by further metabolism of the MEHP.

The majority of available studies indicate that DEHP is without direct genotoxic activity for a variety of genotoxicity endpoints, including mutagenesis, induction of sister-chromatid exchange and dominant lethal effects (Hopkins, 1983). Negative results on chromosomal damage were obtained following treatment of human lymphocytes with DEHP (Turner *et al.*, 1974: Stenchever *et al.*, 1976), of human fetal lung cells (Stenchever *et al.*, 1976) and of Chinese hamster ovary cells (Phillips *et al.*, 1982). DEHP treatment reportedly decreased the mitotic activity and growth rates of human lymphocytes (Turner *et al.*, 1974: Stenchever *et al.*, 1976). Also, DEHP and its metabolites did not appear to bind directly to DNA (Albro *et al.*, 1982a: Kornburst *et al.*, 1984). In addition, DEHP and its metabolites did not appear to affect DNA repair systems or induce clastogenic effects in intact animals exposed to DEHP *in vivo* (Butterworth *et al.*, 1984). Marginal clastogenic effects have been observed in CHO cells in culture (chromosomal aberrations). Embryonic cells (day 12) from Syrian golden hamsters treated on day 11 with 3750 or 15000 mg DEHP/kg or with similar doses of MEHP showed a significant increase in aberrant metaphase cells from all except the lowest treatment groups (Tomita *et al.*, 1982). The high doses utilized in these studies were viewed as confounding factors in the interpretation of the results (Butterworth *et al.*, 1984).

The mutagenic activity of DBP for *Salmonella typhimurium* mutants depended on the assay protocol. In the standard Ames test, DBP was negative in S. typhimurium strains TA98, TA100, TA1535 and TA1537 with and without metabolic activation (Daniel, 1978; Shibko and Blumenthal, 1973). DBP was positive for mutagenicity in a liquid suspension assay with a 4-hour incubation in strain TA100 without metabolic activation and negative with metabolic activation (Seed, 1982). When the Ames test was modified to incorporate histidine and biotin into the bottom agar, DBP was negative with and without metabolic activation in strains TA98 and TA100 (Elder, 1985). DBP was not mutagenic to E. coli or S. cerevisiae (Omori, 1976; Shahin and Von Borstel, 1977). Kawachi et al. (1980) reported that DBP was negative for chromosome aberrations and sister chromatid exchanges (SCEs). Ishidate and Odashima (1977) reported that DBP in a 0.2 percent bovine albumin solution caused a significant increase in chromosome aberrations in Chinese hamster cells. The authors termed DBP a "suspicious compound". DBP in concentrations of 0.25 mg/ml had no effect on chromatid aberrations in human leukocyte cultures (Tsuchiya and Hattori, 1976).

From the available data it would appear that DEHP and DBP are neither mutagenic nor genotoxic, although contradictions in the results obtained with certain test systems diminish the strength of such a conclusion.

20.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

The administration of 1000 mg DEHP/kg body weight to mice on the 7th day of gestation was associated with fetotoxicity: 1260 mg/kg body weight administered between days 1 and 18 of gestation was associated with fetotoxicity and skeletal anomalies: 3420 mg/kg body weight administered between days 1 and 18 of gestation was associated with increased post-implantation mortality: 7200 mg/kg body weight administered between days 1 and 18 of gestation was associated with fetotoxicity and gross abnormalities of the eye and ear (NIOSH, 1982). Teratogenic, embryolethal and fetotoxic effects have been observed in rats treated with DEHP (Peters and Cook, 1973: Singh et al., 1974 & 1975: Nakamura et al., 1979: Oishi and Hiraga, 1980: Fishbein, 1984). Testicular atrophy was observed in rats exposed to 2000 mg DEHP/kg body weight/day in the diet over 14 days (Nixon et al., 1982a). A dose of 20000 mg DEHP/kg body weight/day over 7 days produced seminiferous tubule degeneration and testicular atrophy while 12 500 and 6000 mg/kg body weight produced similar effects over 90 or 730 days of exposure, respectively (Fishbein, 1984). Gangolli (1982) demonstrated that testicular atrophy and depletion of zinc levels in reproductive glands of rats could be produced by treatment with DBP in addition to DEHP. No effects were reported on testicular weight or morphology of marmosets exposed in the diet to 2000 mg DEHP/kg body weight/day for 14 days (Nixon et al., 1982b).

No teratogenic effects were reported in rats or mice at dosage levels not associated with maternal toxicity. Reproductive effects were not observed in marmosets exposed to DEHP. DEHP administration to rats was associated with testicular atrophy, pituitary hypertrophy and depletion of zinc levels in the testis and secondary sex glands. These effects were not observed in mice or marmosets. These data indicate that DEHP, and its hydrolysis product, MEHP, are embryo- and fetotoxic at high levels of exposure and that the incidence of normally occurring developmental abnormalities increased in rats and mice exposed to maternally toxic levels of DEHP. Therefore, DEHP may be prudently regarded as a fetotoxic agent and a potential weak teratogen. However, the lack of reproduction toxicity in other species (rabbits and marmosets) and the high doses required to demonstrate reproductive effects in rats and mice indicate DEHP would not be associated with reproductive effects at levels of exposure near those observed in human populations.

A number of studies have investigated the teratogenicity and reproductive toxicology of DBP; two intraperitoneal and two oral studies are detailed below. Peters and Cook (1973) administered dibutyl phthalate intraperitoneally to pregnant female rats in doses of 2 and 4 ml/kg body weight on days 3, 6 and 9 of gestation (5 rats at each dose regime). DBP administration resulted in a 50% reduction in the number of pups weaned per litter. In addition, two male pups from the 2 ml/kg body weight dosage group were born with no eyes. In another study, groups of 5 pregnant female rats were administered DBP intraperitoneally in doses of 1/3, 1/5, and 1/10 of the IP LD_{50} (3.05 ml/kg body weight). The rats were sacrificed one day before expected parturition. Although there was no effect on fertility, there were significant effects upon embryonic and/or fetal development. In addition there was a significant increase in skeletal abnormalities in the fetuses (Singh et al., 1972). Nikonorow et al. (1973) carried out a gavage study using female rats. Groups of 10 female rats were given 120 and 600 mg/day of DBP in olive oil for three months prior to mating. Separate groups of female rats received the same doses for 21 days following fertilization. Uteri and fetuses from all rats were removed on day 21 of gestation. DBP administration prior to mating did not cause significant changes in the measured parameters. Administration of DBP to pregnant rats resulted in lower placental weights. A significantly increased resorption rate, as well as decreased fetal weights were observed in the 600 mg/day dose group. No adverse effects were reported in the group receiving the 120 mg/day dose. Shiota and Nishimura (1982) administered DBP in the feed of pregnant mice throughout the gestation period at 5 dose levels ranging from 80 to 2100 mg/kg body weight. Resorptions, fetal deaths and maternal toxicity increased with dosage. Further, two of three live fetuses from the 2100 mg/kg body weight group had neural tube defects. In this study, significant effects were not noted below 350 mg/kg body weight/day. From a hazard assessment point of view the oral studies are of greater significance than the intraperitoneal studies. The no-adverse-effect-level of 120 mg/day established by the Shiota and Nishimura (1982) study may be of value in assessing human health hazards associated with DBP exposure. In a review of these and other reproduction and teratology studies Elder (1985) concluded that the available data is not sufficient to conclude that DBP is teratogenic to laboratory animals.

20.4.5. CARCINOGENICITY

DEHP has been shown to be carcinogenic in cancer bioassays in mice and rats (IARC, 1982: Kluwe et al., 1982a: NTPa, 1982: Huff and Kluwe, 1984). Data on male rats were not available, therefore IARC (1982) considered the carcinogenicity data on DEHP equivocal.

Turnbull and Rodericks (1985) assessed the carcinogenic risks associated with DEHP in humans and concluded, on the basis of rodent data, that mechanism of carcinogenicity was most likely related it its ability to induce peroxisome proliferation and other related enzymatic changes. Based on the assumption that the pattern of metabolism is the same in rats as in humans, they predicted a 1 in 10^6 risk of cancer from lifetime of exposure to levels of between 1.5 and 791 ug/kg body weight/day. In this range, a most likely figure was estimated as 116 ug/kg body weight/day.

The differences in metabolism of DEHP between species have raised concerns regarding the validity of extrapolating carcinogenicity data from rats, which cannot glucuronidate the MEHP metabolite of DEHP, to humans, where glucuronidation has been demonstrated (Northup et al., 1982: Kluwe et al., 1983). However, glucuronidation of MEHP does not appear critical to its carcinogenic action since mice have been show to have the ability to conjugate MEHP (Albro et al., 1982a & 1982b) yet develop liver tumours following chronic exposure to DEHP (Kluwe et al., 1982a: NTP, 1982a).

Confounding factors associated with the carcinogenicity bioassay aside, these data do demonstrate that, at high levels of exposure, DEHP was carcinogenic in rats and mice. Utilizing the Squire (1981) ranking system for carcinogens assessed in rodent bioassay systems, DEHP was given a score of 60 (Kluwe et al., 1983), on a scale ranging from 13 (lowest possible score) to 100 (highest possible score, based on knowledge available at the time. This ranking would place DEHP intermediate between such chemical carcinogens as tris(2,3-dibromopropyl) phosphate (score of 90) and DDT (score of 31) (Kluwe et al., 1983). Such ranking of carcinogenic risk associated with chemicals does not consider factors such as the human population size exposed to DEHP, differences in routes of exposure between humans and that used in the animal bioassays and the adequacy of the animal model as a predictor of human carcinogenesis (Kluwe et al., 1983).

The importance of DEHP in the manufacture of plastics and the human health concerns associated with the reported carcinogenicity of DEHP in laboratory animals have resulted in an extensive literature on the mechanism of DEHP induced cancers in laboratory animals and the extrapolation of these observations to humans. The bulk of these data indicate that DEHP induces liver cancer in rodents through non-genotoxic mechanisms.

Dibutyl phthalate is reported to be noncarcinogenic (Kawachi et al, 1980; Abe and Sasaki, 1977; Dernehl, 1973).

20.5. HUMAN TOXICOLOGY

Due to its widespread use in plastics the entire population could potentially be exposed to DEHP. Level of exposure depends on the volatilization from vinyl products and ease of extraction into water. Various phthalate esters are ubiquitous in the environment and have been detected in air, water, soil, vegetation, animal tissues and mineral deposits. It is presumed that these compounds are volatilized on heating or burning of plastics or leach from plastics found in landfills (Thomas and Northup, 1982).

Tissue DEHP levels in autopsy patients ranged from less than 0.3 ug/g wet tissue (detection limit) to 4.6 ug/g (liver). Detectable levels were reported in brain, heart, kidney, liver and spleen. Of nine autopsy patients examined, only 4 had non-detectable levels of DEHP in their tissues (Chen et al., 1979). DBP was found in normal and diseased kidneys, adipose tissue at autopsy, in the blood of pregnant women, and in umbilical cords (Overturf et al, 1978, 1979; Mes et al, 1974). The NTP (1983) estimated that total human exposure from all sources of ingestion was approximately 5.8 mg/day.

Major sources for human exposure are from foods, especially high fat foods, in contact with vinyl plastics and parenteral solutions, often administered intravenously for medical purposes. DEHP can leach from plastic tubing during the pasteurization of milk (Wildbrett, 1973). Particular high exposure groups include patients receiving parenteral fluids, patients undergoing peritoneal dialysis and workers in the plastics industry. Indirect human exposure could occur through drinking of contaminated water or eating of aquatic organisms. DEHP has been detected in various forms of marine life, soil samples and animal and human tissues. Average surface water concentrations of 1 ug/L have been measured.

Milkov et al. (1973) evaluated the health status of 147 workers exposed occupationally to mixtures of phthalate plasticizers (including DBP) for prolonged periods. He identified moderately pronounced toxic polyneuritis in many workers.

A case study is documented in the literature of a chemical worker accidentally swallowing approximately 10 g of DBP. Complete recovery occurred after 2 weeks and symptoms included nausea, vomiting, dizziness, headache, pain and irritation in the eyes, conjunctivitis and toxic nephritis (Krauskopf, 1973).

The cosmetic industry has conducted skin irritation studies for DBP on human subjects. No positive reactions were identified among 53 subjects patch tested with 5% DBP. In another study 1532 human subjects were patch tested with 2% DBP; one positive reaction was observed. Sensitization and photosensitization was not observed (Elder, 1985).

20.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

In 1982, the TLV-TWA for DEHP was 5 mg/m^3 (NIOSH, 1982). The US Environmental Protection Agency regulates DEHP under the Toxic Substances Control Act. The US Food and Drug Administration regulates DEHP as an indirect food additive. No regulatory data were found on DBP.

20.6. HAZARD ASSESSMENT

As DEHP is of greater toxicological significance than DBP and the data base is stronger for DEHP the hazard assessment for phthalate esters will be largely concerned with the latter. The assessment of the potential health hazards associated with exposure to phthalate esters lies in their influence on the procedures utilized in the extrapolation of the carcinogenicity data from laboratory animals to humans. Exposure levels to genotoxic carcinogens associated with acceptable risks (e.g., 1×10^{-6}) of adverse health effects in human populations are usually estimated using mathematical models. However, less conservative methods of estimating exposures (e.g., the use of the safety factor-ADI approach) may be applicable to non-genotoxic carcinogens if such carcinogens show a threshold in their dose-response characteristics. Presumably, the risk of cancer would be substantially reduced at exposure levels below the threshold dose. The designation of a chemical carcinogen as acting through direct genetic versus non-genotoxic mechanisms would have a great impact on the interpretation of the health risks to human populations exposed to the chemical and is the subject of much discussion at present. Phthalate esters, particularly DEHP, represent a prime example of the controversy in this area and, consequently, there are inherent uncertainties in the assessment of the potential health hazards of these chemicals at the present time.

There are substantial species differences in the effects of DEHP on the liver and primate species do not appear to show liver peroxisome proliferation following exposure to DEHP and related compounds. Nonetheless, in the absence of adequate data on the effects of phthalates on the liver in humans, it is assumed that the data from rats on peroxisome proliferation would be applicable to humans exposed to DEHP. Such an assumption would appear to result in a conservative assessment of the potential health hazards associated with exposure to DEHP. It should be stressed that there are no data on which to assess the applicability of this assumption with respect to other phthalate esters.

Based on the above assumptions it is appropriate to accept a NOEL of 60 mg DEHP/kg body weight/day for peroxisome proliferation in rodent liver. From the DBP teratogenicity data a tentative NOEL of 120 mg/day for fetotoxicity/maternal toxicity in pregnant rats was determined. Based on a $1/10^6$ risk of cancer, a daily dose of 116 ug DEHP/kg/day has been suggested in the literature. An ADI of 0-0.6 ug DEHP/kg body weight/day has been suggested (NAS, 1977). The levels of DEHP and DBP present in the Edmonton drinking water were often less than the detection limits (1.0 ug/L) although traces of DBP were reported on several occasions. DEHP was detected at concentrations above the detection limits on several occasions. Although a high level of 7.8 ug/L was reported, concentrations were usually less than 5 ug/L. Given a worst case continuous exposure level of 5 ug/L, intake of 2 L of water in a 60 kg man would result in a daily dose of 0.17 ug/kg body weight/day. As this is lower than either the 10^6 cancer risk, the upper level of the NAS ADI and 360000 fold less than the dose causing peroxisome proliferation in rat liver, it would appear that the health risks associated with exposure to phthalate esters in Edmonton drinking water are negligible.

21. NAME: POLYNUCLEAR AROMATIC HYDROCARBONS

Naphthalene
Anthracene
Phenanthrene

The polynuclear aromatic hydrocarbons are a large and complex group of chemicals, some of which are known to be carcinogenic (Benzo[a]pyrene; 7,12-dimethylbenz[a]anthracene; benz[a]anthracene and 3-methylcolanthrene). Because of variations in the toxicological properties of these chemicals, it is not possible to generalize and treat the entire group for the purposes of assessment. The selected compounds detected in Edmonton drinking water belong to the generic class of polynuclear aromatic hydrocarbons but have not been shown to be carcinogenic. Three of these compounds have similar chemical properties and are therefore discussed as a group.

21.1. CAS Nos.:

Naphthalene	91-20-3
Anthracene	120-12-7
Phenanthrene	85-01-80

21.2. SOURCES AND USES

Naphthalene is a major industrial chemical and is used as a household chemical in the form of moth-balls for protection of woollen garments from damage by cloths-moths. Naphthalene, phenanthrene and anthracene occur in coal tars and crude oils at levels of up to 10%. These and related chemicals are also formed during the incomplete combustion of organic chemicals and are constituents of cigarette smoke. Total polynuclear aromatic hydrocarbon concentrations reported from surface waters range from 0.14 to 2.5 ug/L in the US whereas levels in finished water were in the range of 0.003 to 0.14 ug/L (NAS, 1982). Concentrations of phenanthrene in finished water ranged from 3 to 3300 ng/L (NAS, 1982).

21.3. PHARMACOLOGY AND METABOLISM

Naphthalene is metabolized to 1- or 2-naphthol and 1,2-dihydro-1,2-naphthalenediol in laboratory mammals and man. It is excreted as the glucuronide or sulphate conjugate. Other metabolites such as naphthoquinones and naphthalene oxides have also been reported (Patty, 1981). Phenanthrene is metabolized to 1,2-; 3,4-; and 8,10-dihydrodiols after IP injection in rabbits (Boyland and Wolf, 1950; Boyland and Sims, 1962) and similar metabolites were reported in *in vitro* preparations from guinea pig, mouse and rat livers (Sims, 1970; Charutapit and Holder, 1978 and Nordqvist *et al.*, 1971).

21.4. ANIMAL TOXICOLOGY

21.4.1. ACUTE

The polynuclear aromatic hydrocarbons have comparatively low acute toxicity to mammals. Representative data are given below:

Chemical Form	Species	Sex	Route	Parameter	mg/kg Body Wt.	References
Naphthalene						
	rat	?	IP	LD_{68}	1000	Gosselin, 1984
	man	M	Oral	Lethal dose	5-15 g/person	Patty, 1981
	Child	?	Oral	Lethal dose	2-3 g/child	Patty, 1981
Phenanthrene						
	mouse	?	IP	LD_{50}	700	Simmon *et al.* (1979)

21.4.2. CHRONIC

Chronic oral dosing of rats at 1000 mg phenanthrene/kg body weight for 200 days caused changes in the opacity and shape of the lens of the eye (Gosselin, 1984). Rats injected with 40 mg/kg body weight naphthalene for three days showed inhibition of hepatic arylhydroxylase activity (Clayton and Clayton, 1981).

21.4.3. MUTAGENICITY AND GENOTOXICITY

Tests to date indicate that naphthalene is not a mutagen (Horning *et al.*, 1976). IARC (1983) reported that phenanthrene was mutagenic in only three of the 23 mutagenicity tests conducted on this substance. IARC concluded that there is only limited experimental evidence for the mutagenicity of phenanthrene.

21.4.4. TERATOGENICITY AND REPRODUCTIVE TOXICOLOGY

No information was available in the literature.

21.4.5. CARCINOGENICITY

Naphthalene has been reported to not be carcinogenic (Horning *et al.*, 1976). Phenanthrene was reported to not produce mammary tumors in rats given a single oral dose of 200 mg/kg body weight at age 50 days (Huggins and Yang, 1962). It is not tumorigenic in mouse skin either by SC or topical application with and without initiators such as benzo(a)pyrene and promoters such as croton oil (IARC, 1983). It is also not tumorigenic in neonatal mice injected SC at a does of about 40 mg/kg body weight (Grant and Roe, 1963).

21.5. HUMAN TOXICOLOGY

Exposure to naphthalene causes hemolysis of red blood cells in man, particularly in those of dark skin colour and those with a red-blood cell glucose-6-phosphate dehydrogenase deficiency. No data on human toxicology of phenanthrene was found in the literature.

21.5.1. REGULATORY INFORMATION/EXPOSURE LIMITS/ETC.

No "suggested no adverse response levels" (SNARLs) were developed by the NAS (NAS, 1982), however the World Health Organization (WHO, 1970) has suggested a maximum of 200 ng/L for carcinogenic polynuclear aromatic hydrocarbons in drinking water.

21.6. HAZARD ASSESSMENT

The amount of information on the polynuclear aromatic hydrocarbons is inadequate for an unequivocal hazard assessment. The highest level of naphthalenereported from Edmonton drinking water was <30 ug/L (<30 ppb) while those for anthracene and phenanthrene were consistently <1.0 ppb (1 ug/L). At these concentrations, worst case daily consumption via water in a 60 kg person consuming 2 L of water per day would be as follows:

Phenanthrene	0.03 ug/kg body weight
Anthracene	0.03 ug/kg body weight
Naphthalene	1.00 ug/kg body weight

In the absence of clear evidence of carcinogenicity it is appropriate to use chronic or acute toxic effects as endpoints for hazard assessment. Based on the 40 mg/kg body weight inhibition of rat liver aryl hydroxylase inhibition by naphthalene and, assuming similar activity in anthracene and phenanthrene, these maximum consumptions represent the following risk factors:

Chemical form	Arylhydroxylase inhibition dose/dose in man
Phenanthrene	130000 fold
Anthracene	130000 fold
Naphthalene	40000 fold

The limited toxicological data does not suggest a significant human health risk from exposure to these compounds in Edmonton drinking water.

REFERENCES

Abe S. and M. Sasaki. 1977 Chromosome aberrations and sister chromatid exchanges in Chinese hamster cells exposed to various chemicals. J. Natl. Cancer Inst., 58: 1635-41.

Albro P.W. and B. Moore. 1974 Identification of the metabolites of simple phthalate diesters in rat urine. J. Chromatogr., 94: 209-218.

Albro P.W. and R.O. Thomas. 1973 Enzymatic hydrolysis of di(2-ethylhexyl) phthalate by lipases. Biochem. Biophys. Acta, 360: 380-393.

Albro P.W., I. Tondeur, D. Marbury, S. Jordan, J. Schroeder and J.T. Corbett. 1983 Polar metabolites of di-(2-ethylhexyl) phthalate in the rat. Biochim. Biophys. Acta, 760: 283-292.

Albro P.W., J.R. Hass, C.C. Peck, S.T. Jordan, J.T. Corbett and J. Schroeder. 1982c Applications of isotope differentiation for metabolite studies with di-(2-ethylhexyl) phthalate. J. Environ. Sci. Health, B17: 701-714.

Albro P.W., J.T. Corbett, J.L. Schroeder, S. Jordan and H.B. Matthews. 1982a Pharmacokinetics, interactions with macromolecules and species differences in metabolism of DEHP. Environ. Health Perspect., 45: 19-25.

Albro P.W., S.T. Jordan, J.L. Schroeder and J.T. Corbett. 1982b Chromatographic separation and quantitative determination of the metabolites of di(2-ethylhexyl) phthalate from urine of laboratory animals. J. Chromatogr., 244: 65-79.

Andre F.D., R.L. Buschborn, W.C. Cannon, R.A. Miller and B.D. Hardin. 1981 Teratogenic assessment of ethylbenzene and 2-ethoxyethanol. Battelle Pacific Northwest Laboratories, Richland, WA, Contract PHS-NIOSH-210-79-0037.

Angelo M. 1984 Physiological Pharmacokinetics -- Oral Administration Nutrition Foundation Inc. Food Solvents Workshop I: Methylene Chloride. Bethesda, Maryland, March 8 - 9, 127-135.

Angerer J. and G. Lehnert. 1979 Occupational chronic exposure to organic solvents. VIII. Phenolic compounds - Metabolites of alkylbenzenes in man. Simultaneous exposure to ethylbenzene and xylenes. Int. Arch. Occup. Environ. Health, 43: 145-150.

Ariyoshi T., M. Eguchi, Y. Muraki, H. Yasumatsu, N. Suetsugu and K. Arizono. 1981 Effects of chlorinated benzenes on the activities of sigma-aminolevulinic acid synthetase and heme oxygenase and on the content of hemoprotein in the liver of rats. J. Pharm. Dyn., 4: 69-76.

Autian J. 1973 Toxicity and health threats of phthalate esters: review of the literature. Environ. Health Perspect., 4: 3-26.

Autian J. 1982 Antifertility effects and dominant lethal assays for mutagenic effects of DEHP. Environ. Health Perspect., 45: 115-118.

Avigan J., H.J. Quastel and P.G. Scholefield. 1955 Studies of fatty acid oxidation. 3 -- The effects of acyl-CoA complexes on fatty acid oxidation. Biochem. J., 60: 329-334.

Bardodej Z. and E. Bardodejova. 1970 Biotransformation of ethylbenzene, styrene and alpha-methylstyrene in man. Amer. Ind. Hygiene Assoc. J., 31: 206-209.

Bartsh H., C. Malaveille, A. Barbin and G. Planche. 1979 Mutagenic and alkylating metabolites of halo-ethylenes, chlorobutadienes and dichlorobutenes produced by rodent or human liver tissues. Evidence for oxirane formation by P450-linked microsomal monooxygenases. Arch. Toxicol., 41: 249-277.

Baslo A. and M. Aksoy. 1982 Neurological abnormalities in chronic benzene poisoning. A study of six patients with aplastic anemia and two with preleukemia. Environ. Res., 27: 457-465.

Bedford P.G.C. and E.G.C. Clarke. 1971 Suspected benzoic acid poisoning in the cat. Vet. Rec., 88: 599-601.

Beliles R. P., D. J. Brusick and F. J. Mecler. 1980 Teratogenic mutagenic risk of workplace contaminants: trichloroethylene, perchloroethylene and carbon disulphide. Contract No. 210-77-0047. Litton Bionetics, Inc., Kensington, Maryland.

Bell F.P. 1982 Effects of phthalate esters on lipid metabolism in various tissues, cells and organelles in mammals. Environ. Health Perspect., 45: 41-50.

Bicchi C., M. Mashaly and P. Sandra. 1984 Constituents of Essential Oil of *Nepeta nepetella*. Planta Med., 50:96-98.

Bjorkhem I., Blomstra R. and L. Svensson. 1978 Quantitative importance of omega-oxidation of fatty acids. J. Lipid Res., 19: 585-590.

Blair A., P. Decoufle, and D. Grauman. 1979 Causes of death among laundry and dry cleaning workers. American Jrnl. of Public Health, 69: 508-511.

Bolt H.M. and B. Link. 1980 Zur Toxikologie von Perchlorathylen. Verh. Dtsch. Ges. Arbeitsmed., 20: 463-470.

Bonnet P., G. Raoult and D. Gradiski. 1979 Lethal concentration 50 of main aromatic hydrocarbons. Arch. Mal. Prof. Med. Trav. Secur. Soc., 40: 805-810 (abstract).

Bonse G., T. Urban, D. Reichert and D. Henschler. 1975 Chemical reactivity, metabolic oxirane formation and biological reactivity of chlorinated ethylenes in the isolated perfused rat liver preparation. Biochem. Pharmacol. 24: 1829-1834.

Bornschein R.L., L. Hastings and J. Manson. 1980 Behavioral toxicology in the offspring of rats following maternal exposure to dichloromethane. Toxicol. Appl. Pharmacol., 52: 29.

Boyland E. and G. Wolf. 1950 Metabolism of polycyclic compounds. 6. Conversion of phenanthrene into dihydroxydihydrophenanthrenes. Biochem. J., 47: 64-69.

Boyland E. and P. Sims. 1962 Metabolism of polycyclic compounds. The metabolism of phenanthrene in rabbits and rats: Dihydro-dihydroxy compounds and related glucosiduronic acids. Biochem. J., 84: 571-582.

Brewster D.C., R.S. Jones and D.V. Parke. 1977b Metabolism of cyclohexylate in rat. Biochem. J., 164: 595.

Brewster D.C., R.S. Jones and D.V. Parke. 1977a The metabolism of cyclohexanecarboxylic acid in the isolated perfused rat liver. Xenobiotica, 7: 601-609.

Bronzetti G., C. Corsi and R. Nieri. 1980 Use of the D_7 strain of S. cerevisiae in the determination of the environmental risk. I. Genetic effects of trichloroethylene. Boll. Soc. Ital. Biol. Sper., 56: 1315-1321.

Brown D.M., P.F. Langley, D. Smith and D.C. Taylor. 1974 Metabolism of chloroform. I. The metabolism of ^{14}C-chloroform by different species. Xenobiotica, 4: 151-163.

Brown D.P. and S.D. Kaplan. 1985 U.S. Dept. of Health and Human Services Retrospective Cohort Mortality Study of Dry Cleaner Workers Exposed to Perchloroethylene (draft), April.

Bruckner J.V. and R.G. Peterson. 1976 Evaluation of toluene toxicity, utilizing the mouse as an animal model of human solvent abuse. Abstr. 713. Pharmacology, 18: 244.

Bruckner J.V. and R.G. Peterson. 1978 Effect of repeated exposure of mice and rats to concentrated toluene and acetone vapors. Abstr. 328. Toxicol. Appl. Pharmacol., 45: 359.

Buben J.A. and E.J. O'Flaherty. 1985 Delineation of the role of metabolism in the hepatoxicity of trichloroethylene and perchloroethylene: A dose-effect study. Toxicol. Appl. Pharmacol., 78: 105-122.

Burek J.D., K.D. Nitschke, T.J. Bell, D.L. Wackerle, R.C. Childs, J.E. Beyer, D.A. Dittenber, L.W. Rampy and M.J. McKenna. 1984 Methylene chloride: a two-year inhalation toxicity and oncogenicity study in rats. Toxicology Research Laboratory, Health and Environmental Sciences, Dow Chemical Company, Midland, MI, October 11.

Burkhalter J. and R.L. Balster. 1979 Behavioral teratology evaluation of chloroform in mice. Neurobehavioral Toxicol., 1: 199-205.

Butler T.C. 1961 The reduction of carbon tetrachloride in vivo and reduction of carbon tetrachloride to chloroform in vivo by tissues and tissue constituents. J. Pharmacol. Exp. Ther., 134: 311-319.

Butterworth B.E., E. Bermudez, T. Smith-Oliver, L. Earle, R. Cattley, J. Martin, J.A. Popp, S. Strom, R. Jirtle and B. Mirchalopoulos. 1984 Lack of genotoxic activity of di(2-ethylhexyl) phthalate (DEHP) in rat and human hepatocytes. Carcinogenesis, 5: 1329-1335.

Callen D.F., C.R. Wolf and R.M. Philpot. 1980 Cytochrome P-450 mediated genetic activity and cytotoxicity of seven halogenated aliphatic hydrocarbons in *Saccharomyces cerevisiae*. Mutat. Res., 77:55.

Cantor K.P., R. Hoover, T.J. Mason and L.J. McCabe. 1977 Association of cancer mortality with halomethanes in drinking water. J. Natl. Cancer Inst., 61: 979-985.

Capel I.D., H.M. Dorrell, M. Jenner, M.H. Pinnock and D.C. Williams. 1979 The effect of chloroform ingestion on the growth of some murine tumors. Eur. J. Cancer, 15: 1485-1490.

Caprino L., G. Togna and M. Mazzei. 1976 Toxicological studies of photosensitizer agents and photodegradable polyolefins. Eur. J. Tox., 9: 99-103.

Carlson B.P. and R.G. Tardiff. 1976 Effect of chlorinated benzenes on the metabolism of foreign organic compounds. Toxicol. Appl. Pharmacol., 36: 383-394.

Carlsson A. and T. Lindqvist. 1977 Exposure of animals and man to toluene. Scand. J. Work Environ. Health, 3: 135-143.

Carpenter C.P., C.S. Weil and H.F. Smyth. 1953 Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs and dogs. Arch. Ind. Hyg. Occup. Med., 8: 219-226.

Carpenter C.P., E.R. Kinkead, D.L. Geary, Jr., L.J. Sullivan and J.M. King. 1975 Petroleum hydrocarbon toxicity studies. V. Animal and human response to vapors of mixed xylenes. Toxicol. Appl. Pharmacol., 33: 543-558.

Carter J.E., D.B. Roll and R.V. Petersen. 1974 The in vitro hydrolysis of di(2-ethylhexyl) phthalate for rats, guinea pigs and dogs. Drug. Metab. Dispos., 2: 341-344.

Cater B.R., M.W. Cook, S.D. Gangolli and P. Grasso. 1977 Studies on dibutyl phthalate-induced testicular atrophy in the rat: effect on zinc metabolism. Toxicol. Appl. Pharmacol., 41: 609-618.

Charutapit S. and G.M. Holder. 1978 Studies on the hepatic microsomal metabolism of ^{14}C-phenanthrene. Biochem. Pharmacol., 27: 1865-1871.

Chemical Industry Institute of Toxicology (CIIT). 1978 Two-year-old vapor inhalation toxicity study with toluene in Fischer-344 albino rats: Six-month status summary. Docket No. 42838. 2 pp.

Chen W.S., J. Kerkay, K.H. Pearson, E.P. Paganini and S. Nakamato. 1979 Tissue bis(2-ethylhexyl)phthalate levels in uremic subjects. Anal. Lett., 12: 1517-1535.

Chin B.H., J.A. McKelvey, L.J. Calisti, S.J. Kozbelt and L.J. Sullivan. 1980 A comparison of in vivo and in vitro (tissue explant) techniques: Metabolic profile of ethylbenzene in the rat and the dog. Bull. Environ. Contam. Toxicol., 25: 241-245.

Chiou W.L. 1975 Quantitation of hepatic and pulmonary first-pass effects and its implication in pharmacokinetic study. I. Pharmacokinetics of chloroform in man. J. Pharmac. Biopharmaceut., 3: 193-201.

Clayton G.D. and F.E. Clayton, eds. 1981 Patty's Industrial Hygiene and Toxicolgoy, 3rd Edition. Wiley-Interscience, New York.

Cohen E.N. and W. Hood. 1969 Application of low-temperature autoradiography studies of the uptake and metabolism of volatile anesthetics in the mouse. Anesthesiology, 30: 306-314.

Colacci A., G. Arfellini, M. Mazzullo, G. Prodi and S. Grilli. 1985 Genotoxicity of 1,1-dichloroethane. Res. Comm. Chem. Path. Pharm., 49: 243-254.

Costa A.K. and K.M. Ivanetich. 1980 Tetrachloroethylene metabolism by the hepatic microsomal cytochrome P-450 system. Biochem. Pharmacol., 29: 2863-2869.

Costa A.K. and K.M. Ivanetich. 1982 The 1,2-dichloroethylenes: Their metabolism by hepatic cytochrome P-450 in vitro. Biochem. Pharmacol., 31: 2093-2102.

Costa A.K. and K.M. Ivanetich. 1984 Chlorinated ethylenes: Their metabolism and effect on DNA repair in rat hepatocytes. Carcinogenesis, 5: 1629-1636.

Cronkite E., J. Bullis, T. Inoue and R. Drew. 1984 Benzene inhalation produces leukemia in mice. Toxicol. Appl. Pharmacol., 75: 358-361.

Crump D., A.A. Swigar, J.R. West, R.B. Silverstein, D. Muller-Schwarze and R. Altieri. 1984 Urine fractions that release flehmen in black-tailed deer, Odocoileus hemionus columbianus. J. of Chemical Ecology, 10: 203-215.

Daniel J.W. 1963 The metabolism of ^{36}Cl-labelled trichloroethylene and tetrachloroethylene in the rat. Biochem. Pharmacol., 12: 795-802.

Daniel J.W. 1978 Toxicity and metabolism of phthalate esters. Clin. Toxicol., 13: 257-268.

Dean B.J., T.M. Brooks, G. Hodson-Walker and D.H. Hutson. 1985 Genetic toxicology of 41 industrial chemicals. Mutat. Res., 153: 57-77.

Deichmann W., W. MacDonald and E. Bernal. 1963 The hemopoietic tissue toxicty of benzene vapors. Toxicol. Appl. Pharmacol., 5: 201-224.

Dekant W., M. Metzler and D. Henschler. 1986 Identification of S-1,2,2-trichlorovinyl-N-acetylcysteine as a urinary metabolite of tetrachloroethylene -bioactivation through glutathione conjugation may explain its nephro-carcinogenicity. J. Biochem. Toxicol. in press.

Dernehl C.U. 1973 Toxicity of phthalates via ingestion. Tech. Pap. Reg. Tech. Conf. Soc. Plast. Eng. Palisades Sect. 71-2, March 20-22.

DeSalva S., A. Volpe, G. Leigh and T. Regan. 1975 Long-term safety studies of a chloroform-containing dentrifice and mouth rinse in man. Food Cosmet. Toxicol., 13: 529-532.

Deuel H.J., R. Alfin-Slater, C.S. Weil and H.F. Smyth, Jr. 1954 Sorbic acid as a fungistatic agent for foods. 1 - Harmlessness of sorbic acid as a dietary component. Food Res., 19: 1-12.

Divincenzo G.D. and W.J. Krasavage. 1974 Serum ornithine carbamyl transferase as a liver response test for exposure to organic solvents. Am. Ind. Hyg. Assoc. J., 35: 21-29.

Dobrokhotov V. and M. Enikeev. 1977 Mutagenic effect of benzene, toluene and a mixture of these hydrocarbons in a chronic experiment. Gig. Sanit., 41: 32-34.

Docks E.L. and G. Krisna. 1976 The role of glutathione on chloroform-induced hepatotoxicity. Exp. Mol. Pathol., 24: 13-22.

Dorfmueller M.A., S.P. Henne, R.G. York, R.L. Bornschein and J.M. Manson. 1979 Evaluation of teratogenicity and behavioral toxicity with inhalation exposure of maternal rats to trichloroethylene. Toxicology, 14: 153-166.

Driesbach R.H. 1980 Handbook of Poisoning: Prevention, Diagnosis, and Treatment, Tenth Edition. Lange Medical Publications, Los Altos, California.

Drill V.A. and P. Lazar, eds. 1977 Cutaneous Toxicity, Academic, New York.

Durden D.A. and A.A. Boulton. 1982 Identification and distribution of phenylacetic acid in the brain of the rat. J. Neurochem., 38: 1532-1536.

Elder R.L., ed. 1985 Safety Assessment of Cosmetic Ingredients: Final report on the safety assessment of dibutyl phthalate, dimethyl phthalate, and diethyl phthalate. J. Am. College Tox., 4: 267-303.

Engstrom K., K. Husman and J. Rantanen. 1976 Measurement of toluene and xylene metabolites by gas chromatography. Int. Arch. Occup. Environ. Health., 36: 153-160.

Engstrom K., K. Husman and V. Riihimaki. 1977 Percutaneous absorption of m-xylene in man. Int. Arch. Occup. Environ. Health, 39: 181-189.

Environment Canada (EC). 1984 Benzene. Environmental and Technical Information for Problem Spills, Technical Services Branch, Environmental Protection Service, Ottawa.

Environment Canada (EC). 1984 Ethylbenzene. Environmental and Technical Information for Problem Spills, Technical Services Branch, Environmental Protection Service, Ottawa.

Environment Canada (EC). 1984 Xylenes. Environmental and Technical Information for Problem Spills, Technical Services Branch, Environmental Protection Service, Ottawa.

Environmental Protection Agency (EPA). 1980 Investigation of Selected Potential Environmental Conaminants: Styrene, Ethylbenzene and Related Compounds. EPA Office of Toxic Substances, Washington, D.C., EPA 560/11-80-018.

Environmental Protection Agency (EPA). 1981 Advisory opinion of dichloromethane. Office of Drinking Water, U.S. EPA, Washington DC, Draft.

Environmental Protection Agency (EPA). 1984 Health Assessment Document for Tetrachloroethylene, Research Triangle Park, N.C. pp 8-1 to 8-28.

Environmental Protection Agency (EPA). 1985 Health Assessment Document for Chloroform. EPA-600/8-84-004F. Office of Health and Environmental Assessment, Research Triangle Park, N.C.

Evans I.A. and M.A. Osman. 1974 Carcinogenicity of bracken and shikimic acid. Nature (London), 250: 348.

Feldstein D. and N.C. Klendshoj. 1954 The determination of cyanide in biological fluids by microdiffusion analysis. J. Lab. Clin. Med., 44: 166.

Fernandez J., E. Guberan and J. Caperos. 1976 Experimental human exposures to tetrachloroethylene vapor and elimination in breath after inhalation. Am. Ind. Hyg. Assoc. J., 37: 143-150.

Filser J.G. and H.M. Bolt. 1979 Pharmacokinetics of halogenated ethylenes in rats. Arch. Toxicol., 42: 123-136.

Fishbein L. 1984 Toxicity of the components of poly(vinylchloride) polymers additives. Industrial Hazards of Plastics and Synthetic Elastomers, pp 113-136.Published by Alan R. Liss, NY.

Florin I., L. Rutberg. M. Curvall and C.R. Enzell. 1980 Screening of tobacco smoke constituents for mutagenicity using the Ames's Test. Toxicology, 18: 219-232.

Food and Drug Administration (FDA). 1981 Cosmetic product formulation data: (a) ingredients used in each product category and (b) number of brand name products in each product code. December 22, Washington DC.

Frantz S.W. and P.G. Watanabe. 1983 Tetrachloroethylene: Balance and tissue distribution in male Sprague-Dawley rats by drinking water administration. Toxicol. Appl. Pharmacol., 69: 66-72.

Freitag D., L. Ballhorn, H. Geyer and F. Korte. 1985 Environmental hazard profile of organic chemicals. Chemosphere, 14: 1589-1616.

Friedlander B.R., F.T. Hearne and S. Hall. 1978 Epidemiologic investigations of employees chronically exposed to methylene chloride. J. Occup. Med., 20: 657.

Fry J., T. Taylor and D.F. Hathaway. 1972 Pulmonary elimination of chloroform and its metabolite in man. Arch. Int. Pharmacodyn., 196: 98-111.

Fulton T.R., T. Triano, A. Rabe and Y.H. Loo. 1980 Phenylacetate and the enduring behavioral deficit in experimental phenylketonuria. Life Sci., 27: 1271-1281.

Gangolli S.D. 1982 Testicular effects of phthalate esters. Environ. Health Perspect., 43: 77-84.

Garcha G., P.R. Imrie, E. Marley and D.V. Thomas. 1985 [^{14}C]-beta-phenethylamine, its distribution after administration by various routes to cats, and the effects of monoamine oxidase inhibitors. Br. J. Pharmac., 86: 877-888.

Gerarde H.W. 1959 Toxicological studies on hydrocarbons. III. The biochemorphology of the phenylalkanes and phenylalkenes. A.M.A. Arch. Ind. Health, 19: 403-418.

Gerarde H.W. and D.B. Ahlstrom. 1966 Toxicologic studies on hydrocarbons XI. Influence of dose on metabolism of mono-n-alkyl derivatives of benzene. Tox. Appl. Ph., 9: 185.

Goldstein B.D., C.A. Snyder, A. Laskin, I. Bromberg and R. Albert. 1982 Myelogenous leukemia in rodents inhaling benzene. Toxicol. Lett., 13: 169-173.

Gollamudi R., H.R. Prasanna, R.H. Rao, W.H. Lawrence and J. Autian. 1983 Impaired metabolism of di(2-ethylhexyl) phthalate (DEHP) in old rats -- an in vitro study. J. Toxicol. Environ. Health, 12: 623-632.

Gosselin E.G. 1984 Clinical Toxicology of Commercial Products. Williams and Wilkins, Baltimore, Md.

Grant G. and F.J.C. Roe. 1963 The effect of phenanthrene on tumour induction by 3,4-benzopyrene administered to newly born mice. Br. J. Cancer, 17: 261-265.

Green T. and J. Odum. 1985 Structure/activity studies of the nephrotoxic and mutagenic action of cysteine conjugates of chloro-and fluoroalkenes. Chem.-Biol. Interact., 54: 15-31.

Greenlee W., E. Gross and R. Irons. 1981 Relationship between benzene toxicity and disposition of ^{14}C-labelled benzene metabolites in the rat. Chem.-Biol. Interact., 33: 285-299.

Groger W.K.L. and T.F. Grey. 1979 Effect of chloroform on the activities of liver enzymes in rats. Toxicology, 14: 23-28.

Gromiec and Piotrowski. 1984 Urinary mandelic acid as an exposure test for ethylbenzene. Int. Arch. Occup. Environ. Health, 55: 61-72.

Gueldner R.C., D.A. Nordlund, W.J. Lewis, J.E. Thean and D.M. Wilson. 1984 Kairomones and their use for management of entomophagous insects. XV. Identification of several acids in scales of Heliothis zea moths and comments on their possible role as kairomones for Trichogramma pretiosum. J. of Chemical Ecology, 10: 245-251.

Hardin B.D. and J.M. Manson. 1980 Absence of dichloromethane teratogenicity with inhalation exposure in rats. Toxicol. Appl. Pharmacol., 52: 22.

Harper C., R.T. Drew and J.R. Fouts. 1974 Benzene and p-xylene: a comparison of inhalation toxicities and in vitro hydroxylations. Pp. 302-311 in D.J. Jollow, J.J. Kocsis, R. Snyder, and H. Vainio, eds., Biological Reactive Intermediates: Formation, Toxicity, and Inactivation. Plenum Press, New York.

Hayden J.W., R.G. Peterson and J.V. Bruckner. 1977 Toxicology of toluene (methylbenzene): a review of current literature. Clin. Toxicol., 11: 549-559.

Hazelton Laboratories America Inc. 1981 24-Month chronic toxicity and oncogenicity study of methylene chloride in rats. Submitted to National Coffee Association New York, August.

Hazelton Laboratories America Inc. 1983 24-Month oncogenicity study of methylene chloride in mice. Submitted to National Coffee Association New York, August.

Hearne F.T. and B.R. Friedlander. 1981 Follow-up of methylene chloride study. J. Occup. Med., 23:660.

Henschler D. 1977 Metabolism of chlorinated alkenes and alkanes as related to toxicity. J. Environ. Pathol. Toxicol., 1: 125-133.

Henschler D., E. Eder, T. Neudecker and M. Metzler. 1977 Carcinogenicity of trichloroethylene: fact or artifact? Toxicol., 37: 233-236.

Henschler D., W. Romen, H.M. Elsasser, D. Reichert, E. Eder and Z. Radwan. 1980 Carcinogenicity study of trichloroethylene by longterm inhalation in three animal species. Arch. Toxicol., 43: 237-248.

Heywood R., R.J. Sortwell, P.R.B. Noel, A.E. Street, D.E. Prentice, F.J.C. Roe, P.F. Wadsworth, A.N. Worden and N.J. Van Abbe. 1979 Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol., 2: 835-851.

Hobara T., H. Kobayashi, Higashihara, T. Kawamota and T. Sakai. 1983 Experimental studies of uptake, elimination and metabolism of tetrachloroethylene in dogs by exposure to it. Japan J. Ind. Health, 25: 367-374.

Hofmann H.T., H. Birnstiel and P. Jobst. 1971 Zur inhalationstoxicitatvon 1,1- und 1,2-dichlorathan. Arch. Toxikol., 27: 244.

Hogan M.D., P. Chi, D.G. Hoel and T.J. Mitchell. 1979 Association between chloroform levels in finished drinking water supplies and various site-specific cancer mortality rates. J. Environ. Pathol. Toxicol., 2: 873-887.

Holloway B.R., J.M. Thorp, G.D. Smith and T.J. Peters. 1982a Analytical subcellular fractionation and enzymic analysis of liver homogenates from control and clofibrate treated rats, mice and monkeys with reference to the fatty acid oxidising enzymes. Ann. NY Acad. Sci., 386: 453.

Holloway B.R., M. Bentley and J.M. Thorp. 1982b Species differences in the effects of ICI55897 on plasma lipids and hepatic peroxisomes. Ann. NY Acad. Sci., 386: 439.

Hopkins J. 1983 Is diethylhexyl phthalate genotoxic? Fd. Cosmet. Toxicol., 21: 684-687.

Horning M.G., C.D. Kary, P.A. Gregory and W.G. Stillwel. 1976 Recycling of naphthalene and naphthalene metabolites through the monooxygenase system. Toxicol. Appl. Pharmacol., 37: 118.

Hudak A.K., I. Rodics, G. Stuber, G. Ungvary, G. Krasznai, I. Szomolanyi and A. Csonka. 1977 The effects of toluene inhalation on pregant CFY rats and their offspring. Munkavedelem, 23(1-3, Suppl.): 25-30.

Huff J.E. and W.M. Kluwe. 1984 Phthalate esters carcinogenicity in F344/N rats and B6C3F1 mice. Industrial Hazards of Plastics and Synthetic Elastomers, pp 137-154. Alan R. Liss, Inc, NY.

Huggins C. and N.C. Yang. 1962 Induction and extinction of mammary cancer. Science, 137: 257-262.

IARC. 1979 IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Halogenated Hydrocarbons. International Agency for Research on Cancer, Lyon, France, pp. 545-572.

IARC. 1982 IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 29, International Agency for Research on Cancer, Lyon, France.

IARC. 1983 Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 32 - Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data. International Agency for Research on Cancer, Lyon, France.

Ignat'ev A.D. 1965 Experimental data on the hygienic characteristics of certain chemical food preservatives. Vop. Pitan., 24: 61-68.

Ikeda M. 1977 Metabolism of trichloroethylene and tetrachloroethylene in human subjects. Environ. Health Perspect., 21: 239-245.

Ikeda M. and H. Ohtsuji. 1972 A comparative study of the excretion of Fujiwara reaction-positive substances in urine of humans and rodents given trichloro- or tetrachloro-derivatives of ethane and ethylene. Brit. J. Ind. Med., 29: 99-104.

Infante P., R. Rinsky, J. Wagoner and R. Young. 1977 Leukemia in benzene workers. Lancet, July 9, pp. 76-78.

Inoue K. 1975 Studies on occupational toluene poisoning. (2) An animal experiment using inhalation of toluene vapor in mice. Osaka Shiritsu Daigaku Igaku Zasshi, 24: 791-803.

International Labour Organization (ILO). 1980 Occupational Exposure Limits for Airborne Toxic Substances, Second (Revised) Edition, Geneva, Switzerland.

International Technical Information Institute (ITII). 1981 Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal with Toxicity and Hazard Data. Japan.

Ishidate M. and S. Odashima. 1977 Chromosome tests with 134 compounds on Chinese hamster cells in vitro - A screening for chemical carcinogens. Mutat. Res., 48: 337-53.

Ishidate M. Jr., T. Sofuni, K. Yoshikawa, M. Hayashi, T. Nohmi, M. Sawada and A. Matsuoka. 1983 Primary mutagenicity screening of food additives currently used in Japan. Fd. Chem. Toxic., 22: 623-636.

Jackh R., C. Rhodes, P. Grasso and J.T. Carter. 1984 Genotoxicity studies on di-(2-ethylhexyl) phthalate and adipate and toxicity studies on di-(2-ethylhexyl) phthalate in the rat and marmoset. Fd. Cosmet. Toxicol., 22: 151-155.

Jorgenson T.A., E.F. Meierhenry, C.J. Rushbrook, R.J. Bull and M. Robinson. 1985 Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female $B6C3F_1$ mice. Fund. Appl. Toxicol., 5: 760-769.

Katz R.M. and D. Jowett. 1981 Female laundry and dry cleaning workers in Wisconsin: A mortality analysis. American Jrnl. of Public Health, 71: 305-307.

Kawachi T., T. Yahagi, T. Kada, Y. Tazima, M. Isidate, M. Sasaki and T. Sugiyama. 1980 Cooperative program on short-term assays for carcinogenicity in Japan. IARC Sci. Publ., 27: 323-330.

Kikuchi T., S. Kadota, H. Suehara, A. Nishi, K. Tsubaki, H. Yano and K. Harimaya. 1983 Odourous Metabolites of Fungi, *Chaetomium globosum* KINZE EX FR. and *Botrytis cinerea* PERS. EX FR., and a Blue-green Alga, *Phormidium tenue* (MENEGHINI) GOMONT. Chem. Pharm. Bull., 31: 659-663.

Kimura E.T, D.M. Ebert and P.W. Dodge. 1971 Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol., 19: 699-704.

Kirby P.E., R.F. Pizzarello, T.E. Lawlor, S.R. Haworth and J.R. Hodgson. 1983 Evaluation of di(2-ethylhexyl) phthalate and its major metabolites in the Ames test and L5178Y mouse lymphoma mutagenicity assay. Environ. Mutagenesis, 5: 657-663.

Kirschman J. 1984 National Coffee Assn.'s Testing Program on Methylene Chloride. Nutrition Foundation Inc. Food Solvents Workshop I. Methylene Chloride. Bethesda, Maryland, March 8-9, 26-32.

Kline S.A., E.C. McCoy, H.S. Rosenkranz and B.L. Van Duren. 1982 Mutagenicity of chloroalkene epoxides in bacterial systems. Mutat. Res., 101: 115-125.

Kluwe W.M., E.E. McConnell, J.E. Huff, J.K. Haseman, J.F. Douglas and W.V. Hartwell. 1982a Carcinogenicity testing of phthalate esters and related compounds by the National Toxicology Program and the National Cancer Institute. Environ. Health Perspect., 45: 129-133.

Kluwe W.M., J.K. Haseman and J.E. Huff. 1983 The carcinogenicity of di(2-ethylhexyl) phthalate (DEHP) in perspective. J. Toxicol. Environ. Health, 12: 159-169.

Kluwe W.M., J.K. Haseman, J.F. Douglas and J.E. Huff. 1982b The carcinogenicity of dietary di(2-ethylhexyl) phthalate (DEHP) in Fischer 344 rats and B6C3F$_1$ mice. J. Toxicol. Environ. Health, 10: 797-815.

Kornburst D.J., T.R. Barfknecht, P. Ingram and J.D. Shelburne. 1984 Effect of DEHP on DNA repair and lipid peroxidation in rat hepatocytes and on metabolic cooperation in Chinese hamster V-79 cells. J. Toxicol. Environ. Health, 13: 99-116.

Kozumbo W.J., R. Kroll and R.J. Rubin. 1982 Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect., 45: 103-109.

Krauskopf L.G. 1973 Studies on the toxicity of phthalates via ingestion. Environ. Health Perspect., 3: 61-72.

Lake B.G., J.C. Phillips, J.C. Linnell and S.D. Gangolli. 1977 The in vitro hydrolysis of some phthalate esters by hepatic and intestinal preparations from various species. Toxicol. Appl. Pharmacol., 39: 239-248.

Lang H. and K. Lang. 1956 Fate of benzoic acid-C^{14} in the organism. Arch. Exp. Pathol. Pharmakol., 229: 505-512.

Laskin S. and B. Goldstein, eds. 1977 Benzene toxicity: A critical evaluation. J. Toxicol. Environ. Health Suppl., November 2.

Lawlor T., S.R. Haworth and P. Voytek. 1979 Evaluation of the genetic activity of nine chlorinated phenols, seven chlorinated benzenes, and three chlorinated hexanes. Environ. Mutagenesis, 1: 143.

Lawrence W.H., M. Malik, J.E. Turner, A.R. Singh and J. Autian. 1975 Toxicological investigation of some acute, short-term, and chronic effects of administering di-2-ethylhexyl phthalate (DEHP) and other phthalate esters. Environ. Res., 9: 1-11.

Lhuguenot, J.C., A.M Mitchell, G. Milner, E.A. Lock and C.R. Elcombe. 1985 The metabolism of d-(2-ethylhexyl)phthalate (DEHP) and mono(2-ethylhexyl) phthalate (MEHP) in rats: In vivo and in vitro dose and time dependency of metabolism. Toxicol. Appl. Pharmacol., 80: 11-22.

Lijinski W. and H. Garcia. 1972 Skin carcinogenesis tests of hydrogenated derivatives of anthanthrene and other polynuclear hydrocarbons. Z. Krebforsch. Klin. Onkol., 77: 226-230.

Liu Z., C.T. Liu, G. Fu, Y. Xu, S. Sun, Z Chen and A. Li. 1982 Studies on the chemical composition of Qi Li Xiang rose oil. I. Oxygenated portion. Youji Huaxue, 6: 443-446 (abstract)

Loo Y.H., T. Fulton, K. Miller and H.M. Wisniewski. 1980 Phenylacetate and brain dysfunction in experimental phenylketonuria: synaptic development. Life Sci., 27: 1283-1290.

Lutz K. 1979 In vivo covalent binding of organic chemicals to DNA as a quantitative indicator in the process of chemical carcinogenesis. Mutat. Res., 65: 289-356.

Lyapkalo A.A. 1973 Genetic activity of benzene and toluene. Gig. Tr. Prof. Zabol., 17: 24-28.

Lyon J.P. 1976 Mutagenicity studies with benzene. PhD. thesis, University of California, San Francisco. Diss. Abstr., B36: 55370-B.

Maltoni C., B. Conti, G. Cotti and F. Belpoggi. 1983 A multipotential carcinogen. The up-to-date results of the long-term bioassays performed at the Bologna Institute of Oncology. Am. J. Ind. Med., 4: 589-630.

Mangham A.R., J.R. Foster and B.G. Lake. 1981 Comparison of the hepatic and testicular effects of orally administered di(2-ethylhexyl) phthalate and dialkyl phthalate in the rat. Toxicol. Appl. Pharmacol., 61: 205-214.

Mansuy D., P. Beaune, T. Cresteil, M. Lange and J.P. Lerous. 1977 Evidence for phosgene formation during liver microsomal oxidation of chloroform. Biochem. Biophys. Res. Commun., 79: 513-517.

Margard, W. 1978 In vitro bioassay of chlorinated hydrocarbon solvents. Battelle Laboratories. Unpublished proprietary document for Detrex Chemical Industries. (Cited in EPA 1984).

Marquardt P. 1960 Zur Vertraglichkeit der Benzoesaure. Arzneimittel Forsch., 10: 1033.

Matsushita T., Y. Arimatsu, A. Ueda, K. Satoh and S. Nomura. 1975 Hematological and neuro-muscular response of workers exposed to low concentration of toluene vapor. Ind. Health (Japan), 13: 115-121.

McCall S.N., P. Jurgens and K.M. Ivanetich. 1983 Hepatic microsomal metabolism of the dichloroethanes. Biochem. Pharmacol., 32: 207-213.

McCann J.E., E. Choi, E. Yamasaki and B.N. Ames. 1975 Detection of carcinogens as mutagens in the Salmonella/microsome test: assay of 300 chemicals. Proc. Natl. Acad. Sci., 72: 5135-5139.

McConnell G., D.M. Ferguson and C.R. Pearson. 1975 Chlorinated hydrocarbons and the environment. Endeavor, 34: 13-18.

Mes J., D.E. Coffin and D.S. Campbell. 1974 Dibutyl and di(2-ethylhexyl)-phthalate in human adipose tissue. Bull. Environ. Contam. Toxicol., 12: 721-5.

Milkov L.E., M.V. Aldyreva, T.B. Popova, K.A. Lopukhova, Y.L. Makarenko, L.M. Malyar and T.K. Shakhova. 1973 Health status of workers exposed to phthalate plasticizers in the manufacture of artificial leather and films based on PVC resins. Environ. Health Perspec., 3: 175-8.

Miller T.A., D.H. Rosenblatt, J.C. Dcacre et al. 1976 Problem Definition on Potential Environmental Pollutants IV. Physical, Chemical, Toxicological and Biological Properties of Benzene, Toluene, Xylenes; and p-Chlorophenyl Methyl Sulfide, Sulfoxide and Sulfone. U.S. Army Medical Bioengineering Research and Development Laboratory, Fredrick, MD, ADA04035.

Minor J.L. and B.A. Becker. 1971 A comparison of the teratogenic properties of sodium salicylate, sodium benzoate and phenol. Toxicol. Appl. Pharmacol., 19: 373.

Mitoma C., T. Steeger, S.E. Jackson, K.P. Wheeler, J.H. Rogers and H.A. Milman. 1985 Metabolic disposition study of chlorinated hydrocarbons in rats and mice. Drug Chem. Tox., 8: 183-194.

Miyazawa M., H. Maruyama and H. Kameoka. 1984 Essential oil constituents of *Paeoniae radix*. Agric. Biol. Chem., 48: 2847-2849.

Morton T.C. and M.A. Bateman. 1981 Chemical studies on proteinaceous attractants for fruit flies, including the identification of volatile constituents. Aust. J. Agric. Res., 32: 905-916.

Mungikar A.M. and S.S. Pawar. 1976 Hepatic microsomal mixed function oxidase system during toluene treatment and the effect of pretreatment of phenobarbital in adult rats. Bull. Environ. Contam. Toxicol., 15: 198-204.

Murray F.J., B.A. Schwetz, J.G. McBride and R.E. Staples. 1979 Toxicity of inhaled chloroform in pregnant mice and their offspring. Toxicol. Appl. Pharmacol., 50: 515-522.

Nakamura Y., Y. Yagi, I. Tomita and K. Tsuchikawa. 1979 Teratogenicity of di(2-ethylhexyl)phthalate in mice. Toxicol. Lett., 4: 113.

National Academy of Science (NAS). 1977 Drinking Water and Health. Volume 1. Safe Drinking Water Committee. Adv. Center on Toxicol., Natl. Res. Council, Washington DC.

National Academy of Science (NAS). 1980 Drinking Water and Health, Volume 3. Safe Drinking Water Committee. Adv. Center on Toxicol., Natl. Res. Council, Washington DC.

National Academy of Science (NAS). 1982 Drinking Water and Health, Volume 4. Safe Drinking Water Committee. Adv. Center on Toxicol., Natl. Res. Council, Washington DC.

National Academy of Science (NAS). 1983 Drinking Water and Health, Volume 5. Safe Drinking Water Committee. Adv. Center on Toxicol., Natl. Res. Council, Washington DC.

National Cancer Institute (NCI). 1976a Carcinogenesis Bioassay of Trichloroethylene. Carcinogenesis Tech. Rep. Ser. No. 2. DHEW Publ. No. (NIH) 76-802. Bethesda, Md.: U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health.

National Cancer Institute (NCI). 1976b Report on carcinogenesis bioassay of chloroform. PB-264018. Springfield, VA: National Technical Information Service.

National Cancer Institute (NCI). 1977 Bioassay of tetrachloroethylene for possible carcinogenicity CAS No. 127-18-4. NCI-CG-TR-13 DHEW Publ. No. (NIH) 77-813. Department of Health, Education and Welfare, Washington, D.C.

National Cancer Institute (NCI). 1978 Bioassay of 1,1-dichloroethane for possible carcinogenicity. CAS No. 75-34-3. DHEW/PUB/NIH-78-1316, NCI-CG-TR-66. Carcinogen Testing Program, Bethesda, Md.

National Institute for Occupational Safety and Health (NIOSH). 1982 Registry of toxic effects of chemical substances, microfische edition. R.J. Lewies and R.L. Tatken, (eds.). U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Cincinnati, OH.

National Institute for Occupational Safety and Health (NIOSH). 1978 Chloroethanes: Review of Toxicity. Current Intelligence Bulletin 27, U.S. Department of Health, Education and Welfare.

National Toxicology Program (NTP). 1982b Carcinogenesis bioassay of butylbenzylphthalate (CAS No. 117-81-7) in F344 rats and $B6C3F_1$ mice (feed study). NTP Technical Report Series TR No. 213, Research Triangle Park, NC.

National Toxicology Program (NTP). 1982c Carcinogenesis Bioassay Experimental and Status Report, March 1, 1982. Research Triangle Park, N.C: National Toxicology Program.

National Toxicology Program (NTP). 1982a Carcinogenesis bioassay of di-(2-ethylhexyl) phthalate (CAS No. 117-81-7) in F344 rats and $B6C3F_1$ mice (feed study). NTP-80-37, NIH Publication 82-1773, National Toxicology Program, Research Triangle Park, NC.

National Toxicology Program (NTP). 1983 Third Annual Report on Carcinogens. Summary, September. US Department of Health and Human Services, Publich Health Services, NTP 82-330.

National Toxicology Program (NTP). 1986a Toxicology and Carcinogenesis Studies of Dichloromethane (Methylene Chloride) in F344/N Rats and $B6C3F_1$ Mice (Inhalation Studies). U.S. Department of Health and Human Services, National Institutes of Health, Technical Report Series No. 306. Research Triangle Park, NC.

National Toxicology Program (NTP). 1986b Toxicology and Carcinogenesis Studies of Benzene in F344/N Rats and $B6C3F_1$ Mice (gavage studies). U.S. Department of Health and Human Services, National Institutes of Health, Technical Report Series No. 289. Research Triangle Park, NC.

Nelson B.K., B.J. Taylor, J.V. Setzer and R.W. Hornung. 1979 Behavioral teratology of perchloroethylene in rats. J. Environ. Pathol. Toxicol., 3: 233.

Nikonorow M., H. Mazur and H. Piekacz. 1973 Effect of orally administered plasticizers and polyvinyl chloride stabilizers in the rat. Toxicol. Appl. Pharmacol., 26: 253-259.

NIOSH/OSHA (U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health (NIOSH), U.S. Department of Labor, Occupational Safety and Health Administration (OSHA)). 1981 Occupational Health Guidelines for Chemical Hazards, NIOSH Publication No. 81-123.

Nitschke K.D., T.J. Bell, L.W. Rampy and M.J. McKenna. 1983 Methylene chloride: a two year inhalation toxicity and oncogenicity study in rats. Toxicology Research Laboratory, Health and Environmental Sciences, Dow Chemical Company, Midland, MI, October 11.

Nixon J., S.J. Jackson and C. Rhodes. 1982a Bis(2-ethylhexyl) phthalate: a comparative subacute toxicity study in the rat and marmoset. Report No. CTL/P/690 prepared for CEFIC by Central Toxicology Laboratory, Alderley Park, Macclesfield, Cheshire.

Nixon J., S.J. Jackson and C. Rhodes. 1982b Bis(2-ethylhexyl) phthalate: subacute toxicity by the IP route to marmosets. Report No. CTL/R/644 prepared for CEFIC by Central Toxicology Laboratory, Alderley Park, MacClesfield, Cheshire.

Njaga G.D.E. and H.N.B. Gopalan 1982 Cytogenetic effects of the food preservatives sodium benzoate and sodium sulphite on Vicia faba root meristems. Mutation Res., 102: 213-219.

Nordqvist M., D.R. Thakker, K.P. Vyas, H. Yagi, W. Levin, D.E. Ryan, P.E. Thomas, A.H. Conney and D.M Jerina. 1971 Metabolism of chrysene and phenanthrene to bay region diol epoxides by rat liver enzymes. Mol. Pharmacol., 19: 168-178.

Northup S., L. Martis, R. Ulbricht, J. Garber, J. Miripol and T. Schmitz. 1982 Comment on the carcinogenic potential of di(2-ethylhexyl) phthalate. J. Toxicol. Environ. Health, 10: 493-518.

NRC (U.S. National Research Council). 1981 The Alkyl Benzenes. Committee on Alkyl Benzene Derivatives, Board on Toxicology and Environmental Health Hazards, Assemble of Life Sciences, National Academy Press, Washington, D.C.

Ohtsuki T. K. Sato, A. Koizumi, M. Kumai and M. Ikeda. 1983 Limited capacity to humans to metabolize tetrachloroethylene. Int. Arch. Occup. Environ. Health, 51: 381-390.

Oishi S. and K. Hiraga. 1980 Testicular atrophy induced by phthalate acid monoesters: effects on zinc and testosterone concentrations. Toxicology, 15: 197-202.

Omori Y. 1976 Recent progress in safety evaluation studies on plasticizers and plastics and their controlled use in Japan. Environ. Health Perspect., 17: 203-209.

Opdyke D.L.J. 1979 Monographs on Fragrance Raw Materials: Phenylacetaldehyde. Fd. Cosmet. Toxicol., 17: 357-390.

Otson R., D.T. Williams and P.D. Bothwell. 1982 Volatile organic compounds in water at thirty Canadian potable water treatment facilities. J. Assoc. of. Anal. Chem., 65: 1370.

Ott M.G., L.K. Skory, B.B. Holder, J.M. Bronson and P.R. Williams. 1983 Health evaluation of employees occupationally exposed to methylene chloride. Scand. J. Work Environ. Health, 9: 1-38.

Overturf M.L., R.E. Druilhet, J.G. Liehr, W.M. Kirkendall and R.M. Caprioli. 1979 Phthalate esters in normal and pathological human kidneys. Bull. Environ. Contam. Toxicol., 22: 536-42.

Overturf M.L., R.E. Druilhet, W.M. Kirkendall, J.G. Liehr and R.M. Caprioli. 1978 Occurrence of phthalate esters in human kidney. Clin. Res., 26: 781A.

Palmer A.K., A.E. Street, F.J.C. Roe, A.N. Worden and N.J. Van Abbe. 1979 Safety evaluation of toothpaste containing chloroform. II. Long-term studies in rats. J. Environ. Pathol. Toxicol., 2: 821-833.

Panchenko L.F., S.V. Pirozhkov and V.D. Antonenkov. 1985 Subcellular distribution and properties of a clofibrate-induced aldehyde dehydrogenase from rat liver. Biochemical Pharmacology, 34: 471-479.

Patel J.M., C. Harper and R.T. Drew. 1976 Changes in serum enzymes after inhalation exposure of p-xylene in rats. Abstr. 78. Toxicol. Appl. Pharmacol., 37: 124.

Patty 1981 See Clayton and Clayton, 1981.

Paul G.G. and D. Rubenstein. 1963 Metabolism of carbon tetrachloride and chloroform by the rat. J. Pharmacol. Exp. Ther., 141: 141-148.

Peck C.C. and P.W. Albro. 1982 Toxic potential of the plasticizer di-(2-ethylhexyl) phthalate in the context of its disposition and metabolism in primates and man. Environ. Health Perspect., 45: 11-17.

Peck C.C., D.G. Odom, H.J. Freidman, P.W. Albro, J.R. Hass, J.T. Brad and D.A. Hess. 1979 Di-2-ethylhexyl phthalate (DEHP) and mono-2-ethylhexyl phthalate (MEHP) accumulation in whole blood and red cell concentrates. Transfusion, 19: 137-146.

Pegg D.G., J.A. Zempel, W.H. Braun and P.G. Watanabe. 1979 Disposition of tetrachloro(^{14}C)ethylene following oral and inhalation exposure of rats. Toxicol. Appl. Pharmacol., 51: 465-474.

Perocco P., S. Bolognesi and W. Alberghini. 1985 Toxic activity of seventeen industrial solvents and halogenated compounds on human lymphocytes cultured in vitro. Toxicol. Letters, 16: 69-75. .

Peters J.W. and R.M. Cook. 1973 Effect of phthalate esters on reproduction in rats. Environ. Health Perspect., 3: 91.

Phillips B.J., T.E.B. James and S.D. Gangolli. 1982 Genotoxicity studies of di(2-ethylhexyl) phthalate and its metabolites in CHO cells. Mutat. Res., 102: 297-304.

Poll W.E. 1963 Skin as a test site for the bioassay of carcinogens and carcinogen precursors. The First International Conference on the Biology of Cutaneous Cancer. Nat. Cancer Inst. Monogr., 10: 611-631.

Potempska A., Y.H. Loo and H.M. Wisniewski. 1984 On the possible mechanism of phenylacetate neurotoxicity: inhibition of choline acetyltransferase by phenylacetyl-CoA. J. Neurochem., 42: 1499-1501.

Reid W.D., B. Christie, G. Krishna, J.R. Mitchell, J. Moskowitz and B.B. Brodie. 1971 Bromobenzene metabolism and hepatic necrosis. Pharmacology, 6: 41-55.

Rhudy R.L., J.W. Lindberg, D.J. Goode and E.J. Gralla. 1978 Ninety-day subacute inhalation study with toluene in albino rats. Toxicol. Appl. Pharmacol., 45: 284-285.

Rinsky R.A., R.T. Young and A.B. Smith. 1981 Leukemia in benzene workers. Am. J. Ind. Med., 2: 217-245.

Rock G., V.E. Secours, C.A. Franklin, I. Chu and D.C. Villeneuve. 1978 The accumulation of mono-2-ethylhexyl phthalate (MEHP) during storage of whole blood and plasma. Transfusion, 18: 553-558.

Rodkey S.L. and H.R. Collison. 1977a Biological oxidation of ^{14}C-methylene chloride to carbon monoxide and methane by rats. Toxicol. Appl. Pharmacol., 40: 33.

Rodkey S.L. and H.R. Collison. 1977b Effect of dihalogenated methane *in vivo* production of carbon monoxide and methane by rats. Toxicol. Appl. Pharmacol., 40: 39.

Rowland I.R. 1974 Metabolism of di(2-ethylhexyl)phthalate by the contents of the alimentary tract of the rat. Fd. Cosmet. Toxicol., 12: 293-302.

Rowland I.R., R.C. Cottrell and J.C. Phillips. 1977 Hydrolysis of phthalate esters by the gastrointestinal contents of the rat. Fd. Cosmet. Toxicol., 18: 17-21.

Rozen M.G. and C.A. Snyder. 1985 Protracted exposure of C57BL/6 mice to 300 ppm benzene depresses B- and T-lymphocyte numbers and mitogenic responses. Toxicology, 37: 13-26.

Rozenbaum N.D. et al. 1947 Use of chlorobenzene as a solvent from the standpoint of industrial hygiene. Gig. Sanit., 12:21.

Ruddick J.A., D.C. Villeneuve, I. Chu, E. Nestmann and D. Miles. 1981 An assessment of the teratogenicity in the rat and mutagenicity in *Salmonella* of mono-2-ethylhexyl phthalate. Bull. Environ. Contam. Toxicol., 27: 181.

Sato A., T. Kakamima, Y. Fujiwara and K Hirosawa. 1974 Pharmacokinetics of benzene and toluene. Int. Arch. Arbeitsmed., 33: 169-182.

Sax, N.I., ed. 1984 Dangerous Properties of Industrial Materials, Sixth Edition. Van Nostrand Reinhold, New York.

Schachter D. 1957 The chemical estimation of acyl glucuronides and its application to studies on the metabolism of benzoate and salicylate in man. J. Clin. Invest., 36: 297-302.

Schumann A.M., J.F. Quast and P.G. Watanabe. 1980 The pharmacokinetics and macromolecular interactions of perchloroethylene in mice and rats as related to oncogenicity. Toxicol. Appl. Pharmacol., 55: 207-219.

Schwetz B.A., B.K.J. Leong and P.J. Gehring. 1974a Embryo- and fetotoxicity of inhaled chloroform in rats. Toxicol. Appl. Pharmacol., 28: 442-451.

Schwetz B.A., B.K.J. Leong and P.J. Gehring. 1974b Embryo- and fetotoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone in rats. Tox. Appl. Pharm., 28: 452-464.

Schwetz B.A., B.K.J. Leong and P.J. Gehring. 1975 The effect of maternally inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol., 32: 84-96.

Sedivec V. and J. Flek. 1976 The absorption, metabolism, and excretion of xylenes in man. Int. Arch. Occup. Environ. Health, 37: 205-217.

Seed L.J. 1982 Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect., 45: 111-114.

Shahin M.M. and R.C. Von Borstel. 1977 Mutagenic and lethal effects of alpha-benzene hexachloride, dibutyl phthalate and trichloroethylene in Saccharomyces cerevisiae. Mutat. Res., 48: 173-80.

Shelton D.W. and L.J. Weber. 1981 Quantification of the joint effects of mixtures of hepatotoxic agents: Evaluation of a theoretical model in mice. Environ. Res., 26: 33-41.

Shibko S.I. and H. Blumenthal. 1973 Toxicology of phthalic-acid esters used in food packaging material. Environ. Health Perspec., 3: 131-7.

Shimada T., A.F. Swanson, P. Leber and G.M. Williams. 1985 Activities of chlorinated ethane and ethylene compounds in the Salmonella/rat microsome mutagenesis and rat hepatocyte/DNA repair assays under vapor phase exposure conditions. Cell Biol. and Toxicol., 1: 159-219.

Shiota K. and H. Nishimura. 1982 Teratogenicity of di(2-ethylhexyl)-phthalate (DEHP) and di-n-butyl phthalate (DBP) in mice. Environ. Health Perspect., 45: 65-70.

Simmon V.F., H.S. Rosenkranz, E. Zeiger and L.A. Poirier. 1979 Mutagenic activity of chemical carcinogens and related compounds in the intraperitoneal host-mediated assay. J. Natl. Cancer Inst., 62: 911-918.

Simmon V.F., K. Kauhanen and R.B. Tardiff. 1977 Mutagenic activity of chemicals identified in drinking water. In: Progress in Genetic Toxicology, D. Scott, B.A. Bridges and F.H. Sabels, eds. Elsevier, NY. pp. 249-258.

Simmon V.F., K. Kauhanern, K. Mortelmans and R. Tradiff. 1978 Mutagenic activity of chemicals identified in drinking water. Mut. Res., 53: 262, Abst. No. 191.

Sims P. 1970 Qualitative and quantitative studies on the metabolism of a series of aromatic hydrocarbons by rat-liver preparations. Biochem. Pharmacol., 19: 795-818.

Singh A.R., W.H. Lawrence and J. Autian. 1972 Teratogenicity of phthalate esters in rats. J. Pharm. Sci., 61: 51-5.

Singh A.R., W.H. Lawrence and J. Autian. 1974 Mutagenic and antifertility sensitivities of mice to di-2-ethylhexyl phthalate (DEHP) and dimethoxyethyl phthalate (DMEP). Toxicol. Appl. Pharmacol., 29: 35.

Singh A.R., W.H. Lawrence and J. Autian. 1975 Dominant lethal mutations and antifertility effects of di-2-ethylhexyl adipate and diethyl adipate in male mice. Toxicol. Appl. Pharmacol., 32: 566.

Slacik-Erben R., R. Roll, G. Franke and H. Uehleke. 1980 Trichloroethylene vapours do not produce dominant lethal mutations in male mice. Arch. Toxicol., 45: 37-44.

Slater G.P. and V.C. Blok. 1983 Isolation and indentification of odourous compounds from a lake subject to cyanobacterial blooms. Wat. Sci. Tech., 15: 229-240.

Snyder R., S.L. Longacre, D. Sammett, C.M. Witmer and J.J. Kocsis. 1983 Relationship between the toxicity and metabolism of benzene. Adv. Mod. Environ. Toxico., 4: 23-26.

Sodemoto Y. and M. Enomoto. 1980 Report on carcinogenesis bioassay of sodium benzoate in rats: Absence of carcinogenicity of sodium benzoate in rats. J. Environ. Pathol. Toxicol., 4: 87-95.

Spector W.S., ed. 1956 Handbook of Toxicology, Volume 1. Saunders, Philadelphia and London.

Squire R.A. 1981 Ranking animal carcinogens: a proposed regulatory approach. Science, 214: 211-215.

Stenback F. and P. Shubik. 1974 Lack of toxicity and carcinogenicity of some commonly used cutaneous agents. Tox. and App. Pharm., 30: 7-13.

Stenchever N.A., M.A. Allen, L. Jerominski and R.V. Peterson. 1976 Effects of bis(2-ethylhexyl) phthalate on chromosomes of human leukocytes and human fetal lung cells. J. Pharmacol. Sci., 65: 1648-1651.

Stewart R.D. and H.C. Dodd. 1964 Absorption of carbon tetrachloride, trichloroethylene, tetrachloroethylene, methylene chloride and 1,1,1-trichloroethane through the human skin. Am. Ind. Hyg. Assoc. J., 25: 439-446.

Stewart R.D., H.H. Gay, D.S. Erley, C.L. Hake and A.W. Schaffer. 1961 Human exposure to tetrachloroethylene vapor. Relationship of expired air and blood concentrations to exposure and toxicity. Arch. Environ. Health, 2: 516-522.

Stott W.T., J.F. Quast and P.G. Watanabe. 1982 The pharmacokinetics and macromolecular interactions of trichloroethylene in mice and rats. Tox. and App. Pharm., 62: 137-151.

Tahti H., J. Ruusha and H. Vapaatalo. 1977 Toluene toxicity studies on rats after one week inhalation exposure. Acta Pharmacol. Toxicol. Suppl., 41: 78.

Takeuchi Y. and N. Hisanaga. 1977 The neurotoxicity of toluene: EEG changes in rats exposed to various concentrations. Br. J. Ind. Med., 34: 314-324.

Tanaka A., A. Matsumoto and T. Yamaha. 1978 Biochemical studies of phthalate esters. III. Metabolism of dibutyl phthalate (DBP) in animals. Toxicology, 9: 109-123.

Tanii H. and K. Hashimoto. 1985 Studies on the mechanism of acute toxicity of nitriles in mice. Arch. Toxicol., 55: 47054.

Theiss J., G. Stoner, M. Shimkin and E. Weisburger. 1977 Test for carcinogenicity of organic contaminants of United States drinking water by pulmonary tumor response in strain A mice. Cancer Res., 37: 2717-2720.

Thomas J.A. and S.J. Northup. 1982 Toxicity and metabolism of monoethylhexylphthalate and diethylhexylphthalate: A survey of recent literature. J. Toxicol. Environ. Health, 9: 141-152.

Thompson D.J., S.D. Warner and V.B. Robinson. 1974 Teratology studies on orally administered chloroform in the rat and rabbit. Toxicol. Appl. Pharmacol., 29: 348-357.

Tola S., R. Vilhunen, E. Jarvinen and M.L. Korkala. 1980 A cohort study on workers exposed to trichloroethylene. J. Occup. Med., 22: 737.

Tomasi A., E. Albano, A. Bini, B. Botti, T. Slater, F. Trevor and V. Vannini. 1984 Free radical intermediates under hypoxic conditions in metabolism of halogenated carcinogens. Toxicol. Pathol., 12: 240-246.

Tomita I., Y. Nakamura, N. Aoki and N. Inui. 1982 Mutagenic/carcinogenic potential of DEHP and MEHP. Environ. Health Perspect., 45: 119-125.

Tove S.B. 1964 Toxicity of saturated fat. J. Nutr., 84: 237-243.

Tsuchiya K. and K. Hattori. 1976 Chromosomal study on human leukocyte cultures treated with phthalic acid ester. Hokkaidoritsu Eisei Kenkyusho Ho, 26: 114.

Turnbull D. and J.V. Rodericks. 1985 Assessment of possible carcinogenic risk to humans resulting from exposure to di(2-ethylhexyl) phthalate (DEHP). J. Am. Coll. Toxicol., 4: 111-145.

Turner J.H., J.C. Petricciani, M.L. Couch and S. Wenger. 1974 An evaluation of the effects of diethylhexyl phthalate (DEHP) on mitotically capable cells in blood packs. Transfusion, 14: 560-566.

Uehleke H., T. Werner, H. Greim and M. Kramer. 1977 Metabolic activation of halothane and tests in vitro for mutagenicity. Xenobiotica, 7: 393-400.

Ungvary G., A. Hudak, Z. Bors and G. Folly. 1976 The effect of toluene on the liver assayed by quantitative morphological methods. Exp. Mol. Pathol., 25: 49-59.

Van Dyke R.A., M.B. Chenoweth and A. van Poznak. 1964 Conversion in vivo of several anesthetics to $^{14}CO_2$ and chloride. Biochem. Pharmacol., 13: 1239-1247.

Verrett M.J., W.F. Scott, E.F. Reynaldo, E.K. Alterman and C.A. Thomas. 1980 Toxicity and teratogenicity of food additive chemicals in the developing chicken embryo. Toxicol. Appl. Pharmacol., 56: 265-273.

Vierstra R.D. and K.L. Poff. 1981 Mechanism of specific inhibition of phototropism by phenylacetic acid in corn seedling. Plant Physiol., 67: 1011-1015.

Vigliani E. 1976 Leukemia associated with benzene exposure. Ann. N.Y. Acad. Sci., 271: 143-151.

Wahlberg J.E. 1976 Percutaneous toxicity of solvents. A comparative investigation in the guinea pig with benzene, toluene, and 1,1,2-trichloroethane. Ann. Occup. Hyg., 19: 115-119.

Wallace C.J. 1959 Hepatitis and nephrosis due to cough syrup containing chloroform. Calif. Med., 73: 442.

Waskell L. 1978 A study of the mutagenicity of anesthetics and their metabolites. Mutat. Res., 57: 141-153.

Watanabe G.I. and S. Yoshida. 1970 The teratogenic effect of benzene in pregnant mice. Acta Med. Biol. (Niigata), 17: 285-291.

Weisenberger B.L. 1977 Toluene habituation. J. Occup. Med., 19: 569-570.

Weiss G. 1980 Hazardous Chemicals Data Book, Environemental Health Review Number 4. Noyes Data Corp., Park Ridge, NJ.

Weitberg A.B., S.A. Weitzman, E.P. Clark and T.P. Stossel. 1985 Effects of antioxidants on oxidant-induced sister chromatid exchange formation. J. Clin. Invest., 75: 1835-1841.

Wen G.Y., H.M. Wisniewski, J.W. Shek, Y.H. Loo and T.R. Fulton. 1980 Neuropathology of phenylacetate poisoning in rats: an experimental model of phenylketonuria. Ann. Neurol., 7: 577-566.

Westrick J.J., J.W. Mello and R.F. Thomas. 1984 The groundwater supply survey. Am. Water Works Assoc., 76: 52-9.

Wheeler A.W., V. Gil and A.J. MacLeod. 1980 Phenylacetonitrile; an auxin-like growth substance from sugar beet (*Beta vulgaris*). J. Sci. Food Agric., 31: 243-246.

White R.D., D.L. Earnest and D.E. Carter. 1983 The effect of intestinal esterase inhibition on the *in vivo* absorption and toxicity of di-n-butyl phthalate. Fd. Cosmet. Toxicol., 21: 99-101.

Wildbrett G. 1973 Diffusion of phthalic acid esters from PVC milk tubing. Environ. Health Perspect., 3: 29-35.

Williams R.T. 1959 Detoxication Mechanisms. Chapman and Hall, London.

Windholz M., ed. 1983 The Merck Index. Merck & Co., Inc., Rahway, N.J.

Winneke G. and G.G Foder. 1976 Dichloromethane produces narcotic effect. Int. J. Occup. Health Saf., 45: 34-35, 49.

Withey J. 1984 Classical Pharmacokinetics -- Oral Administration Nutrition Foundation Inc. Food Solvents Workshop I: Methylene Chloride. Bethesda, Maryland March 8-9, 135-149.

Wolf M., V. Rowe, D. McCollister, R. Hollingsworth and F. Oyen. 1956 Toxicological studies of certain alkylated benzenes and benzene. AMA Arch. Ind. Health, 14: 387-398.

World Health Organization (WH0). 1983 Benzoic acid and its calcium, potassium and sodium salts. FAO/WHO 27th Meeting. Technical Report No. 18.

World Health Organization (WHO). 1970 European Standards for Drinking-Water, 2nd edition. World Health Organization, Gevena, Switzerland.

World Health Organization (WHO). 1984 Guidelines for Drinking-Water Quality, Volume 2. Health Criteria and Other Supporting Information. Geneva, Switzerland.

Yang R.S.H., R.E. Peterson and J.M. Fujimoto. 1979 Increased bile-duct pancreatic fluid flow in benzene and halogenated benzene-treated rats. Toxicol. Appl. Pharmacol., 47: 505-514.

Yllner S. 1961 Urinary metabolites of ^{14}C-tetrachloroethylene in mice. Nature (London), 191: 820.

Yoshikawa K., A. Tanaka, T. Yamaha and H. Kurata. 1983 Mutagenicity study of nine nonalkyl phthalates and dialkyl phthalates using Salmonella typhimurium and Escherichia coli. Fd. Cosmet. Toxicol., 21: 221-223.

Zeiger E., S. Haworth, W. Speck and K. Mortelmans. 1982 Phthalate esters testing in the National Toxicology Program's Environmental Mutagenesis Test Development Program. Environ. Health Perspect., 45: 99-101.

Review of Trace Organics Monitoring

a task report for

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

by

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June 1986

Review of Trace Organic Monitoring of Edmonton Water by the City of Edmonton's Rossdale Water Treatment Plant Laboratory.

1. Basis for Opinions.

The opinions expressed in this report are the result of a visit by R.T. Coutts to the Rossdale Water Treatment Plant on February 17, 1986; an interview with the Lab. Scientist responsible for the monitoring of organic compounds in water; a tour of the laboratory facilities; and a review of documentation on trace organic monitoring provided by the City of Edmonton through Dr. S.E. Hrudey.

2. Organics Monitoring Program.

In the Rossdale laboratory, relatively little emphasis is placed on the analysis of organic compounds in water samples. Only volatile organics in water are analyzed on a routine basis. The procedure used is the 'purge and trap' method, followed by GC analysis of the trapped components. An external standard is added to permit a semi-quantitative estimate of the amounts of components present in the sample. The identities of these components are deduced from their retention times on the GC column, and by comparisons of these times with those of reference (authentic) samples treated identically. The compounds that are identified in this way are mainly halogenated hydrocarbons, and the test is applied daily to treated water samples.

This analytical procedure would be unacceptable in superior laboratories, and requires improvement. Identification of compounds based only on GC retention times is not a sound analytical technique. At least on occasions, identifications should be confirmed by a mass spectrometric technique. The technique used for quantification also needs improvement. Capillary column gas chromatography should be used and mixtures containing known quantities of authentic volatile compounds should be routinely prepared and analyzed by the 'purge and trap' procedure to confirm the accuracy of the analytical technique.

A 'head space' analytical procedure for volatile organics is currently being introduced. There appears to be some initial problems with the procedure, especially with high blank (control) values, but this problem can be solved. The introduction of capillary GC to analyze the 'head space' volatiles is planned.

The experience at the Alberta Environmental Centre in Vegreville is interesting in this regard. The 'head space' procedure is rarely employed in the laboratories in Vegreville. It has proven to be less reliable than the 'purge and trap' method, and difficulties have been encountered with blank values. It is the opinion of the staff at Vegreville that the 'purge and trap' method is a superior, and more reproducible procedure for analysing volatile compounds.

Quantitative analyses of various organic compounds in raw and treated water are required for the AWWA Survey and are performed once a year only at the Rossdale Plant. Comments on this service are to be found on page 5.

3. Use of Commercial Laboratories.

It is also the current practice at the Rossdale plant to engage commercial laboratories to analyze water samples for the organic compounds they obtain, other than volatile organics, if the need arises. Some of the analytical results supplied by the commercial laboratories used (Can-Test Ltd., Enviro-Test Laboratories, Kinetic Environmental Research Laboratories) are of limited value. They are of little help in the identification of compounds other than those which commonly occur in water samples or those which are included in reference libraries. An evaluation of the results supplied by commercial laboratories can be found on pages 5-8.

The suggestion was made that if it is necessary to employ commercial laboratories to identify and quantify organic compounds in water samples, a "cocktail" of known organic compounds should first be prepared in the Rossdale laboratory, and send it to prospective commercial laboratories for identification and quantification of components. Such a procedure would certainly confirm the reliability of available analytical services and reveal whether these services are necessary.

Pesticides and herbicides in Edmonton water are monitored only once a year. Samples are sent to commercial laboratories for analysis and results have not always been satisfactory. The testing for these compounds should be performed more often at appropriate times of year when the chances are that they could be present in river water in significant quantities. These tests could be performed at the Rossdale laboratory if it were expanded and more experienced personnel were made available.

4. Frequency of Sampling at the Rossdale Plant.

Raw water samples and water samples from the distribution system are not regularly analysed for their organic components. More emphasis should be placed on this aspect of quality control. Raw water samples are never removed at night from the N. Saskatchewan River for analysis. This implies that waste materials, including organics, could be dumped in the river overnight and escape detection. Water samples (raw water, water in the distribution system, and treated water) should be collected for analysis at various times during a 24 hr period. Such a procedure would help identify sources of pollution. In summary, times of sampling; frequency of analyses; number of compounds monitored; and analytical techniques all require considerable revision.

5. Record Keeping.

Analytical records that were available for inspection were generally incomplete. They lacked details on the analytical procedures used, and did not include raw analytical data. For example, GC traces containing the actual measurements of peak heights or areas were lacking for the separated organics in water samples and in prepared reference mixtures of authentic compounds. In the absence of such information, a meaningful assessment of analytical expertise and results cannot be made. Better records must be kept so that they can be referred to at a later date for comparison purposes, or for assessment for accuracy by an independent authority.

6. Manpower and Expertise.

A serious problem exists at the Rossdale laboratories in the performing of the analytical procedures just described, and that is the question of personnel. It is readily apparent that insufficient staff are assigned to the monitoring of organic compounds in water samples. It is essential that one individual is given the responsibility on a full time basis to organize, develop and supervise the organics program. At present one individual (Mr. Rashid) attempts to do this but he has many other responsibilities and little or no time to perfect analytical procedures, and no qualified or experienced people to assist him to do so. Clearly, no one at the Rossdale laboratories is capable of interpreting in detail the GC/MS results obtained from commercial laboratories. Unless reliable interpretations are made by the commercial laboratory's personnel, their results are of little value. In any case, those results that are obtained are generally received too late, i.e. after the emergency is over. This means that the analytical data received is grossly underutilized by Rossdale personnel. It is filed and, to all intents and purposes, apparently forgotten.

Although the Rossdale laboratory seems to have a good complement of staff, they do not appear to be used to advantage, and there seems to be a lack good supervision of technical staff. When I visited the laboratories at Rexdale, Toronto and at Vegreville, it was clear that most personnel were interested in their work, were capable, experienced and industrious. There was a feeling of excitement and involvement. These are not the impressions I had when I visited the Rossdale laboratories. The staff that I saw seemed to lack enthusiasm and did not convey the impression that they were particularly interested in what they were doing. Morale was said to be low. The expertise of the laboratory personnel requires review. It appears that their expertise has been mainly acquired 'on the job'. This is acceptable if qualified, experienced personnel supervise the training and have the time to do so, but this is not the case. Technical staff require better supervision, and more senior personnel should be involved in the monitoring of raw and treated water, rather than on research topics.

My overall impression is that there is a lack of leadership, a lack of experienced personnel to conduct reliable microanalyses of organic compounds in water samples, and deficiences in knowledge of analytical techniques. There also appears to be little interest by some senior personnel in improving significantly the scope and standards of analytical services offered. This is in great contrast to the approach adopted by other provincial and federal monitoring agencies.

A complete reorganization of the Trace Organics Monitoring Program is clearly necessary. Further comments on this subject are included in my recommendations.

Recommendations

1. The Rossdale laboratories should provide the following analytical services on a routine basis:

   i) Identification and quantification of volatile organics, including halogenated compounds in raw and treated water samples. Such compounds are odorous, may have objectionable tastes, and may be constituents of "spills". Many are toxic compounds and some may be introduced during the purification process. They can be analyzed relatively quickly and relatively easily on a routine basis. Suitable procedures for the analysis of volatiles are the 'purge and trap' method followed by GC analysis, and the 'head space' analytical method, but a combination of 'purge and trap' and capillary GC is preferred. Calibration graphs using known amounts of authentic volatile organic compounds must also be prepared on a regular basis to ensure that the analytical procedure is reproducible and is providing accurate data. Records, and the actual GC traces on which measurements are made must be retained and available for inspection.

   ii) Isolation and GC separation of extractable acidic, basic and neutral organics, but not their identification and quantification. This would quickly reveal whether the water samples contained unacceptable quantities of extractable organics, and, if so, would dictate the level of treatment required. Known quantities of appropriate reference compounds could be routinely added to the water samples to provide some indication of the quantities of acids, bases and neutrals present.

Trained personnel would be required for these tasks. The supervisor should devote most of his/her time to organizing and maintaining an excellent analytical service. The supervisor should have at least an MSc degree in an analytical area of expertise, significant and current experience in extraction and GC techniques, and some practical experience in the analysis of trace amounts (ppb level) of organic compounds in aqueous and other environmental and/or biological samples. Technical assistance would also be required. Technicians should possess at least an appropriate baccalaureate degree or a college education in an analytical area, have experience and be innovative. Technicians who wait to be told what to do are inappropriate. The number of technicians required would depend on the number of water samples that require to be analyzed.

2. A much more elaborate analytical service which would permit the rapid identification and quantification of organic compounds that are present in significant amounts in raw and in treated water is also required. This would be an expensive service because it would require sophisticated instrumentation (e.g. combined GC/MS; HPLC; perhaps combined HPLC/MS), and trained personnel. A service such as that offered by the Ontario Ministry of the Environment would be ideal, but at this point in time, it would be inappropriate to establish such a facility in an expanded Rossdale laboratory. Such a service could be provided by the Alberta Environmental Centre in Vegreville, by Alberta Research Council, by a commercial laboratory, by appropriate laboratories at the University of Alberta, at a later date by a greatly expanded Rossdale laboratory, or by another analytical agency with suitable expertise and experience.

Regardless of where the analyses are conducted, they must be performed by experienced personnel who are able to devise and implement appropriate analytical procedures and, most importantly, interpret results. For example, an expertise in mass spectrometric interpretation is essential if reliable deductions on compound identity are to be made. Of great importance would be the time interval between sample submission and delivery of results. A rapid service is essential if 'sensitive' and potentially dangerous pollutants are to be identified and their sources revealed. Such a service should be readily available when required, i.e. when routine GC analyses in the Rossdale laboratory indicate unacceptably high quantities of volatile organics or extractable acids, bases and neutrals in water samples. A personal impression, based on discussions with Drs. Smillie and Kumar of the Alberta Environmental Centre in Vegreville, is that this service could be offered by the Vegreville laboratory, provided requests were limited to 30-50 water samples per year. Under these circumstances, results could be supplied within 3 weeks of submission; in real emergency situations, results could even be available within 2-3 days.

Analyses for organic pollutants performed at the Rossdale Water Treatment Plant.

(A) Routine analyses for AWWA Survey. Annual reports for 1982, 1983 and 1984 were available for inspection [see documents E8, E9 and E10 supplied by Dr. Hrudey].

Approximately 150 compounds are identified as possible pollutants in the AWWA survey form. In 1982, only 14 compounds were analyzed in raw water (11 once, and 3 twice) and 13 in finished water. None was detected. In 1983, monitoring was performed for 85 compounds in raw and in finished water. For the vast majority of these compounds (81), monitoring was performed on only one occasion. None of the 85 compounds was detected. In 1984, 57 compounds were monitored, on one occasion only, in raw and in finished water. Seven of the compounds were detected, all within regulatory limits.

Records available for inspection were lacking in many details:

1. Was the analytical procedure appropriate?
2. Were compound identifications confirmed unequivocally?
3. What procedure(s) was (were) applied in the quantitative analyses? Were the accuracies of these results confirmed by another analyst?
4. Were the samples collected at an appropriate time such as after a heavy rainfall?

These questions could not be answered from the material included in the records supplied by Dr. Hrudey. Detailed records of analytical procedures and results must be maintained and be available for inspection.

Analyses of pollutants are performed only once a year. A more frequent monitoring program is required.

(B) A summary of accidental spills since 1981, and the analyses performed on water samples at the Rossdale Plant as a result of these spills, using a 'purge and trap'/GC procedure, was examined [see document E21 supplied by Dr. Hrudey]. Few details were provided. It was impossible to judge the quality of these analyses from the data available. Better analytical records must be kept.

Analyses for organic pollutants performed by commercial laboratories.

(C) Enviro-Test Laboratories analyzed 5 water samples from the EL Smith and Rossdale Plants on December 22, 1983 [sample numbers 11-83-622-1, -2, -4, -5, and -6]; another 5 samples on December 19, 1984 [sample numbers 12-84-1182-1 to -5], and a single sample [number 9-84-1075] on September 28, 1984 [see documents E1, E2 and E3 supplied by Dr. Hrudey].

EPA recommended methodologies were used. The samples were extracted and three fractions were isolated for GC and GC/MS analysis. These fractions were base/neutrals, acids, and herbicides/pesticides. A "semi-quantitative" procedure was used to determine the amounts of some organic compounds in the water samples. For pesticides and herbicides, calibration curves were constructed; pure water and spiked water controls were also analyzed. "Total organics" were also measured.

It was recommended highly that the Water Treatment Plant conducted a routine screening of water samples for toluene, xylene and trimethylbenzene, but it is unclear why these three, and only these three compounds were selected for routine monitoring.

Conclusion: The analytical service provided by Enviro-Test Labs is good. Two weakness are a lack of compound identification (except hydrocarbons, and some aromatic compounds) by GC/MS, and omission of the method used for quantitation of identified compounds.

(D) Can-Test Ltd. analyzed 5 water samples and an alum sample on August 31, 1985 [see document E4 supplied by Dr. Hrudey].

EPA methods, similar to those used by Enviro-Test Labs. were employed. Analyses of extractable organic compounds, pesticides and herbicides were performed. Results were summarized but no spectra were provided for examination. The limited amount of material available made it impossible to judge the quality of the results obtained.

(E) Kinetic Environmental Research Labs. analyzed 6 water samples from the EL Smith Plant, collected on April 15, 1982, and 7 samples from the Rossdale Plant, collected on the same day [see documents E5, E6, E7 and E13 supplied by Dr. Hrudey].

The extraction procedures used were EPA-recommended ones. Raw and treated water samples were examined. The GC separations are good. The mass spectra generated were compared to reference and a few compounds were unequivocally identified. Confidence in identifications was scaled from 1-5. Reliance was placed completely on 'best matches' with reference spectra. Little or no deductions of compound identity were made by interpretation of mass spectra from first principles. Such an approach would have indicated that some identities obtained by the 'best match' principle were clearly wrong. A "semiquantitative" procedure was used to quantitate some of the components present. Fuel oil components, ethylbenzene, dimethylbenzenes, 1,3,3-trimethylcyclopropane, chlorophenols and phthalates dominated in raw water samples.

(F) Analyses were conducted by a commercial company (probably Kinetic Environmental Research Labs.) on 4 samples from the EL Smith Plant (March 29, 1982) [sample numbers 7054-7057], on 8 samples from the same Plant (April 23, 1982) [sample numbers 12308-12315], on 4 samples from the Rossdale Plant (March 29, 1982) [sample numbers 7052, 7053, 7058, 7059], and on 8 samples from the Rossdale Plant (April 23, 1982) [sample numbers 12300-12307] [see documents E16-E19 supplied by Dr. Hrudey]. GC/MS identities of organic components in raw and treated water samples were made in much the same way as described above in section (E).

Conclusions applicable to analyses (E) and (F):

1. Identifications of components other than hydrocarbons, are generally poor. Some 'hits' or 'positive identifications' are clearly wrong.
2. No GC or GC/MS runs of authentic reference compounds were made for direct comparisons with extracted compounds.
3. Method of quantitation was not described.
4. Only extractable, GC-volatile compounds get analyzed by the procedure used. What about other organic compounds in the water which are not readily extracted or are relatively non-volatile?

(G) Enviro-Test Labs. analyzed 2 water samples on June 1 and October 30, 1984 after accidental spills. [see document E20 supplied by Dr. Hrudey].

Analytical procedures applied were good, including interpretation of mass spectra. The method used to quantitate extracted components was "semiquantitative", i.e. a known quantity of a "surrogate reference sample" (2-fluorobiphenyl) was added and GC detector responses of this reference compound and the extracted organic compounds were compared to provide a measure of the amounts of compounds in extracts. This is a reasonable analytical approach when an extract contains many compounds, some of which are unidentified.

(H) Can-Test Ltd. performed a GC/MS analysis of a water sample (sample number 4755) [see document E12 supplied by Dr. Hrudey].

Only mass spectra were provided, with no other details. Identities were based entirely on comparisons with a limited number of library spectra. Tentative identities were not confirmed.

(I) A commercial laboratory performed a GC/MS analysis of a water sample (number 3-82-19) [see document E11 supplied by Dr. Hrudey].
No methological details of the analysis were supplied. Identification of components of GC peaks were attempted by using a library of mass spectra, and selecting only the three most probable 'hits' for each spectrum. A large number (155) of compounds were tentatively identified in this way, and some identifications are obviously incorrect. No attempts were made to confirm any of the tentative identifications, or to quantify any of the components. This type of analytical procedure is of little or no value.

(J) Enviro-Test Laboratories analyzed city water samples on seven occasions between September 27, 1984 and March 28, 1985 [see document E29 supplied by Dr. Hrudey].
Report 9-84-1071: Two raw water samples and two treated samples were analyzed for total extractable hydrocarbons. Conclusion:- The method used is satisfactory and quantitative results are acceptable.
Report 9-84-1075: One raw water sample was analyzed for hydrocarbons by head space analysis; extracted and extractable components analyzed and quantified by GC/FID; major extracted components were characterized by GC/MS. Conclusion:- Details are lacking, but the conclusion that the contaminants are straight and branched hydrocarbons is reasonable. The quantitative procedure used produces only very approximate ('ballpark') results.
Report 2-85-1271: This water sample is claimed to contain various aromatic compounds including polycyclic aromatics (e.g. anthracene, phenanthrene). No mass spectra were provided, so accuracy of compound identity is not known. Approximate quantities of each identified component were determined. Conclusion:- At best, these results are very approximate.
Report 3-85-1296-2: EL Smith water sample was analyzed by purge and trap analysis and by GC/MS. Identities of four major components were deduced to be 2-methylpropanal, 2- and 3-methylbutanal, and chloroform. Conclusion:- I agree with the deductions made.
Report 3-85-1296: A water sample from EL Smith was analyzed for acidic components. The major components were identified by GC/MS as butyric acid, cyclohexanecarboxylic acid, benzoic acid and phenylacetic acid, but they were present only in trace amounts (<3 ppb). Conclusion:- I agree with the proposed identifications of these components.
Report 3-85-1300: A raw EL Smith water sample was analyzed for extractable base/neutral and acidic components. Both groups of compounds were present in very low concentrations (<5 ppb). Conclusion:- The proposed identities of the major extractable acids are correct (cf. report 3-85-1296).
Report 3-85-1302: A water sample was analyzed for volatile organics by the purge and trap procedure. The volatile compounds identified were claimed to be the same as those described in reports 2-85-1271 and 3-85-1296-2, but no details were supplied in support of this claim.
Overall Conclusion: With regard to the material in document E29, I have confidence in the qualitative identifications made and I am reasonably satisfied with the quantitative data . The analytical procedure used, however, is limited to the analysis of organic compounds which can be extracted and for which reference mass spectra are available for comparison. Compounds of unknown structures could not be identified or quantified, and compounds which are not extracted will escape detection when the described analytical procedures are used.

(K) Reports of Water Treatment Plants [1984 Annual Report and February 1985 Monthly Report were supplied by Dr. Hrudey].
The 1984 Annual Report reveals (pp. 62-66) that the following tests, and their

frequencies, were performed:

i) Tests for Aldrin & Dieldrin, Carbaryl, Chlordane, DDT, Diazinon, Diclofop-Methyl, Endrin, Heptachlor, Hexane, Lindane, MCPA, Methoxychlor, Methylparathion, Parathion, PCBs, Pentane, Total Pesticides, Toxaphene, Treflan, Triallate, 2,4-D, 2,4,5-TP, 2-Bromo-1-chloropropane - 5 tests were performed for these compounds during 1984.

ii) Benzene, methyl chloride - 8 tests were performed for these compounds during 1984.

iii) Ethylene dibromide, o-xylene - 4 tests were performed for these compounds in 1984.

iv) Miscellaneous: 161 tests for phenols; nine tests for toluene; 585 tests for total organic carbon; 17 tests for trichlorophenol; 567 tests for trihalomethanes; 565 tests for volatile organics were performed during 1984.

v) (p. 77) Ten reservoirs were monitored once per month for total organic carbon. In 1985, 12 reservoirs (identified) were monitored for total organic carbon [see February 1985 Report, Table 2.3.1]. Amounts varied from 1.80-3.95 mg/L. Rossdale water was worst.

The 1984 average levels of phenols (<0.002 mg/L), organic carbon (2.22 mg/L) and trihalomethanes (<0.001 mg/L) are reported on p. 67. The tests identified above revealed that, of the organic compounds monitored (only 17 are identified on pp. 79-82), not once were levels above recommended limits.

In the February 1985 report, 22 organic compounds are listed in Table 2.2.1 which identifies those tests to be performed on water samples. Only one of them (phenol) was measured (5 times), and it was within acceptable concentration limits.

(L) A document entitled "Enclosure II. What are we trying to treat?" [see document E14 supplied by Dr. Hrudey] presumably complements document E13. In document E14, reference is made to 19 grab samples which were submitted to a commercial laboratory for GC/MS analysis. The analyses conducted were limited to volatile, extractable organic compounds. High molecular weight, more complex compounds were not analyzed; purgeable compounds and pesticides were not analyzed because of high cost. Library searches and comparisons suggested identities of 103 compounds of which 28 were 'very confident' identifications. Quantitative analyses of some compounds were made, but no details of the procedure used were provided. The presence of fuel oil components (hydrocarbons), aromatic compounds, chlorinated hydrocarbons and chlorinated phenols in the samples was claimed.

Identies of some of the identified compounds are equivocal. Confirmatory analyses of authentic samples of authentic samples of the identified compounds under identical conditions were not performed.

An approach for future routine analyses is suggested. The approach is a good one in theory, although no details are provided. If an improved analytical procedure is to be introduced, it must be capable of identifying unequivocally the components of raw and treated water. Authentic reference compounds must be available for this purpose. The procedure must also incorporate an acceptable method for the quantitation of major and "sensitive" components.

(M) The Water Treatment Plant is producing a manual of the analytical procedures used for water samples [document E15 supplied by Dr. Hrudey]. Sections 2, 3.18 and 3.26 [Sampling and Sample Preservation; Phenols; Total Organic Carbon (TOC) Analysis, respectively] are of significance to analytical procedures for organic compounds. The methods used in these three sections of the manual are appropriate. They are based on accepted, literature procedures. Analytical procedures for Phenols and TOC, for example, are closely based on procedures described in "Standard Methods for Examination of Water, 15th edition" (1985).

Visit by R.T. Coutts to the Drinking Water Section of the Water Resources Branch in Toronto (February 1986).

Discussions with Dr. K.J. Roberts (Manager, Drinking Water Section) and several of his senior staff (Mr. R. Hunsinger, Mrs. A. Vajdic, Ms. H.M. Tosine, Dr. V.Y. Taguchi, Dr. C.D. Hall, and Dr. R.E. Clement) in Toronto provided useful comments and detailed information on analytical programs conducted by the Drinking Water Section.

Brief Comments

1. Dr. Ken Roberts, Manager, Drinking Water Section, Water Resources Branch, Ontario Ministry of the Environment, has between 40 and 50 scientific personnel responsible to him.

2. The Drinking Water Section is responsible for the analysis of drinking water samples produced by all water purification plants in Ontario. This amounts to as many as 1500 treatment facilities if very small facilities are included (at many of them only the 'on site' chemistry laboratory tests are applied), but around 500 would be a more reasonable estimate of the facilities that could truly be called treatment plants. Of these, 35 of the largest plants, representing about 70% of the water purification operation, supply water to the Drinking Water Section for routine monitoring. On average, each plant supplies 7.5 samples per year at present, for this purpose.

3. Raw water, treated water and treated water samples from two distribution locations are analyzed.

4. There are about 20 water treatment plants on Lake Ontario, 10-12 of them between Hamilton and Oshawa. Four plants supply metro Toronto, the two major ones being in Mississauga.

5. There are 7 major water treatment plants in the St. Clair - Detroit River area. Spills have occurred in this region and extra monitoring of raw and treated water has been conducted. The continued possibility of spills occurring has led to the introduction of a Drinking Water Surveillance Program (DWSP) by the Ontario Ministry of the Environment. The purpose of this comprehensive program is the provision of immediate, reliable and current information on drinking water quality across Ontario.

6. Very frequent monitoring is obviously possible if the need arises, e.g. after a spill. On one occasion, as a result of a spill, perchloroethylene levels in raw and treated water were monitored twice daily.

7. Chlorine dioxide is not used very much in the treatment of the water samples. It is used sometimes in a few plants, and sometimes when there is a persistent taste and odour problem.

8. Activated charcoal is used intermittantly, but not routinely.

9. Treatment with chlorine is used mainly in conjunction with coagulation with alum, then filtration through a fine filter. It is recognized that treatment with chlorine can produce chlorinated products.

10. In Ontario, there are few problems with hydrocarbons in raw or treated water.

11. Benzene and carbon tetrachloride are only occasionally encountered in water samples.

12. Great care is taken with water sample collection. Stainless steel pumps and stainless steel or copper piping is used in sampling equipment. Sample bottles are of glass. Plastic containers are avoided. All sources of contamination are eliminated.

13. Great care is also taken to ensure that the treated water sample exactly corresponds to the raw water sample from which it was produced. The exact time for the raw sample to progress through the treatment procedure is known.

14. No preservatives are added to the water samples to be analyzed for organic compounds. 'Appropriate ' preservatives are added to water samples other than those tested for organic compounds.

15. Senior personnel: Dr. K.J. Roberts, Manager, Drinking Water Section, Water Resources Branch, Ontario Ministry of the Environment; Mr. R. Hunsinger, Head of Assessment Group; Mrs. A. Vajdic, Head of Policy/Assessment Section; Ms. H.M. Tosine, Manager, Drinking Water Organic Section; Dr. V.Y. Taguchi, Head, Mass Spectrometry Unit; Drs. C.D. Hall, Supervisor, Drinking Water Organics Section; Dr O.W. Berg, Head, Research and Development Unit, Organic Characterization; Dr. R.E. Clement, Supervisor, Dioxin Laboratory; Mr. B. Loescher, Manager, Inorganic Trace Contaminants Section; Mr. L. Vlassoff, Manager, General Inorganic Tests.

Analytical programs conducted by the Drinking Water Section

1. An established monitoring program
2. Introduction of a) a Drinking Water Surveillance Program (DWSP)
   b) a Perchloroethylene Drinking Water Program,
   c) a Dioxin Drinking Water Program
3. Drinking Water Survey St. Clair-Detroit River Area Update January 1986.

1. Established program

The existing analytical program of the Drinking Water Section is undergoing change as the DWSP is introduced. The analytical procedures which are used in the established program for organic compounds are as follows:

i. Purge and Trap.

The trapped, purged volatiles (27 organic compounds) are thermally transferred to a GC containing dual capillary columns, one with an FID and another with an EC detector. The purge/trap/capillary GC system is calibrated daily; methanol is used as the reference standard. Analyses by this method are quantitative. The 27 organic compounds which are analyzed by this procedure are identified in Table I.

ii. GC.

A total of 123 organic compounds including chlorinated organic compounds, chlorinated acid herbicides, organophosphate pesticides and triazine herbicides, carbamates and phenylurea compounds, alachlor, polyaromatics and phenols (Table II), are analyzed quantitatively, mainly by GC, although HPLC is also employed in the analysis of some of the compounds in Table II.

GC is also used to quantify non-volatile, extractable compounds in water samples. The extractable compounds are divided into base/neutrals and acids. The list of base/neutrals which are analyzed contains 43 compounds; the list of acids contains 12 compounds, 11 of which are phenols (Table III).

iii. HPLC.

This chromatographic procedure is used to some extent in the quantitative analysis of some of the 123 compounds identified in Table II.

iv. GC/MS.

There is overlap between the GC and GC/MS analyses.

Combined GC/MS is used routinely to identify and analyze quantitatively the 24 organic compounds identified in Table IV. These compounds are all volatiles and are removed from the water samples by the purge and trap procedure. Two internal reference standards are routinely added to the water samples before purging.

GC/MS is also used to identify and quantify non-volatile extractable organic compounds (base/neutrals and acids) where appropriate, and identify 'unknown compounds' in GC traces.

GC/MS is used routinely to identify any compounds of doubtful or unknown structures. This is achieved by interpretation of their mass spectra from first principles and/or by use of computerized libraries of reference compounds.

Qualitative and quantitative analyses are performed by GC/MS. The list of compounds analyzed by this procedure is dynamic; compounds will be added to the list if the necessity arises.

The Mass Spectrometry Unit in the Drinking Water Section's Rexdale Laboratories has an Envirochem Unacon 810-B Organics Concentrator attached to an HP 5890 GC in sequence with an HP 5970 mass spectrometer. This is the system used to analyze purgeable volatiles. A 60m DB-5 capillary column is employed.

A Finnegan 4000 MS is used to separate, identify and approximately quantify base/neutral and acid extractables. A 30m DB-1 capillary column is used for this purpose. Another older mass spectrometer (make unknown) is also available for occasional use for identification and quantitative analysis of organic compounds in water samples.

2. Drinking Water Surveillance Program

The objectives of this surveillance program are identified in the attachment entitled "Outline of Drinking Water Surveillance Program - January 1986". The DWS Program is designed to include eventually all municipal water supplies in Ontario. Initially, 35 water supply locations have been chosen for surveillance but this number is expected to increase to about 90 locations within four years. The expansion of this DWS surveillance program will, of course, replace existing small monitoring programs.

The Drinking Water Surveillance Program (DWSP) has the following components:

a) Chemistry laboratory tests. [There is also an 'on site' field chemistry laboratory test component].
b) Bacteriological tests.
c) Metal analyses.
d) GC analyses:
    i. volatile organics
    ii. pesticides
    iii. specific pesticides
    iv. chloroaromatics
    v. chlorophenols
    vi. extractable base/neutrals and acids (mainly phenols).
e) HPLC analyses.
f) GC/MS analyses.

Initially, in the DWSP, 139 tests will be performed routinely on each water sample 6 to 10 times a year. The same tests will be applied more often if the situation demands it. During a recent spill situation in the St. Clair-Detroit River area, these 139 tests were performed on a weekly basis.

DWSP Tests:

i) In the DWSP, six field chemistry laboratory tests are performed 'on site': turbidity (sometimes determined more than once daily); chlorine (combined, free and total); pH; temperature (calibrated thermometers supplied). Analysis kits are supplied and standardized techniques are used.

ii) At the Rexdale laboratories, 18 chemistry lab tests are performed: alkalinity, calcium, chloride, color, conductivity, fluoride, hardness, magnesium, sodium, ammonium total, nitrite, nitrate, total nitrogen (Kjeldahl), pH, phosphorus filtered reactive, phosphorus total, total solids, turbidity.

iii) Analysis of metals: 21 tests are performed - aluminum, arsenic, barium, boron, beryllium, cyanide, cadmium, cobalt, chromium, copper, iron, mercury, manganese, molybdenum, nickel, lead, selenium, strontium, uranium, vanadium, zinc. Atomic absorption spectroscopy (AAS) is used for the analyses of metals.

iv) Bacteriological tests: 5 are performed - F. Coliform MF count; standard plate count MF; presence/ absence test for Coliform bacteria; total Coliform MF count; total Coliform background MF count.

v) Analysis of Pesticides: 22 pesticides (identified 'PST') and 18 specific pesticides (identified 'SPC') (i.e. a total of 40 pesticides) are included in these analytical procedures**.

vi) Analysis of Volatile Organics: 29 compounds classified as volatiles (identified 'VOL') are included in the program**. With one exception (1,2-dibromomethane) all the compounds in Table IV are included in this number. Six other volatile compounds not included in Table IV are added, i.e. trans-1,2-dichloroethylene, dichloromethane, dichloroethane, total trihalomethanes, 1,4-dichlorobenzene, 1,3-dichlorobenzene).

vii) Analysis of Chloroaromatics: 14 compounds identified as chloroaromatics (identified 'CHA') are included**.

viii) Analysis of Chlorophenols: Six compounds classified as chlorophenols (identified 'CHP') are included**.

** Summation of v) - viii) shows that a total of 89 organic compounds are routinely qualitatively and quantitatively analyzed for in raw and treated water samples. Identities of all 89 compounds are readily obtained from the text "Drinking Water Survey; St. Clair - Detroit River Area Update January, 1986", published by the Ontario Ministry of the Environment. Most of them are also identified in Chart 2 in "Outline of Drinking Water Surveillance Program January 1986"; see also Table V, attached.

Summary	No. of Tests
i) Field tests 'on site'	6
ii) Inorganic and physical organic tests	18
iii) Metals	21
iv) Bacteriological tests	5
v) - viii) Organics (pesticides, volatiles, chlorinated organics)	89
Total	139

The v) - viii) tests are all conducted gas chromatographically, using various procedures.

Another 19 organic compounds are also analyzed for by combined gas chromatography/mass spectrometry (GC/MS) if required. These 19 compounds are identified in the "Drinking Water Survey St. Clair - Detroit River Area" Report (compounds # 140-158). Other compounds may be added to the GC/MS list. Of the 19 compounds just referred to, four were already included in the v) - viii) list, so the actual number of organic compounds included in the Ontario Drinking Water Surveillance Program is 89 + 15 = 104.

3. Perchloroethylene Drinking Water Program

This was an intensive monitoring program for perchloroethylenes. It began in August 1985 at eight St. Clair River area water treatment plants as a result of an industrial spill, and was terminated at the end of December 1985. Further details on this program can be obtained from "Drinking Water Survey St. Clair - Detroit River Area Update January, 1986".

A weekly monitoring for perchloroethylenes is included in the Drinking Water Surveillance Program referred to above.

4. Dioxin Drinking Water Program.

This program was introduced in November 1985. Water samples from the St. Clair area water treatment plants will be monitored regularly for tetra-, penta-, hexa-, hepta-, and octa-dibenzofurans and -dibenzodioxins. More details on this program are to be found in "Drinking Water Survey St. Clair - Detroit River Area Update January, 1986", pp. 5-7. It is noteworthy that an interim maximum acceptable concentration of 15 parts per quadrillion (ppq) for dibenzodioxins and dibenzofurans in drinking water has been implemented.

In the Dioxin Laboratory, there are three mass spectrometers devoted to the analysis of dioxins in water samples. Two of these instruments (Finnegan models) are low resolution instruments, while one is a high resolution one (12,000 resolution; VG ZAB instrument). All three mass spectrometers are connected to gas chromatographs. The mass spectrometers are operated in total scan and selected ion monitoring (SIM) modes. The low resolution GC/MS scans provide information on groups of dioxins present in the sample. The high resolution instrument is much more sophisticated; it is capable of separating and quantifying all the dibenzodioxin groups, and individual isomers within each group. Some isomers are extremely toxic (e.g. 2,3,7,8-dibenzodioxin), and must be unequivocally identified and quantified.

Conclusion

The Drinking Water Section of the Water Resources Branch in Toronto is an excellent facility. Extremely capable personnel are employed and senior personnel provide the necessary supervision of more junior staff and technicians. The Section is well organized and extremely well equipped. The level of expertise is high as is the quality of the analyses provided.

It was clear from my interviews with various employees in Toronto that the senior personnel were experts in their fields and genuinely interested in providing the best analytical service possible. The qualitative and quantitative results that I examined were first class. I have no criticisms of the Drinking Water Section to offer.

Visit by R.T. Coutts to the Alberta Environmental Centre in Vegreville on June 5, 1986.

The Alberta Environmental Centre in Vegreville provides many services. Its Pollution Control Division has a Water Analytical Services Program and a Trace Organics Analysis and Methods Development and Research Services Program. Discussions with Dr. Dean Smillie, head of the Methods Development and Research Services Group, and Dr. Yogesh Kumar, a senior professional staff member in that Group, were conducted. Dr. Smillie's group is involved in the development and use of sensitive and specific analytical procedures for the identification and quantification of organic compounds present in trace amounts in water samples.

Brief Comments

1. Most of the samples analyzed are those which have undergone purification treatments at various plants in Alberta [two plants in Calgary; two in Edmonton (Rossdale and EL Smith Treatment Plants); and the plants in Drumheller, Fort McMurray, Grande Prairie, Peace River, Lethbridge, Medicine Hat, Red Deer, Olds, Brooks, Camrose, Wetaskiwin and Hinton].

2. Samples of treated water are obtained from each of these treatment plants on a monthly basis. The tests performed on these samples are identified in the 'Treated Water Survey', published annually by the Municipal Engineering Branch, Pollution Control Division, Environmental Protection Services, Alberta Environment.

3. Only treated water samples are generally analyzed, but occasionally some raw water samples, and water samples collected in an Alberta location after a spill or other emergency situation, are also received for analysis.

4. Unlike the Water Resources Branch in Toronto, the Alberta Environmental Centre does not offer a dioxin analytical program. In Alberta, there is no dioxin problem. If a dioxin incident did arise, the analytical procedures currently used at Vegreville would reveal the presence of dioxins. Analytical techniques could be introduced to identify and quantitate them, if required.

5. As well as treated water samples, soil and sediment samples are also analyzed at Vegreville.

Analytical programs conducted by the Pollution Control Division

1. Analysis of water and soil and sediment samples for 'target compounds'. Organic compounds so designated are identified in the attached 'Alberta Environmental Centre Chemical Analysis Request' sheet, and in the 'Treated Water Survey' referred to above. Analytical procedures that are used routinely are now described.
a) A 'purge and trap' procedure is used to remove volatile organic compounds from water samples. The trapped compounds are identified and quantified by combined gas chromatography/mass spectrometry (GC/MS). The 'purge and trap' procedure is preferred to the alternative 'head space' analysis method. The former technique is easier to use, and provides more reliable and reproducible results.
b) Acidic, basic and neutral organic compounds are extracted from water samples using procedures recommended by the Environment Protection Agency (U.S.), and the organic compounds in the extracts are separated, identified and quantified by injection of extracts into gas chromatographs equipped with flame ionization, nitrogen/phosphorus and electron-capture detectors, or chromatographs attached to mass spectrometers.

c) Extractions, attempted identification using MS techniques, and approximate quantification of extractable organic compounds of equivocal or unknown structure are also conducted. In this procedure, 3 or 4 reference standards are added to the water samples in known amounts to assist in the semiquantification of extractable organic compounds of unknown structures. This is an acceptable analytical procedure.

2. Specialized analytical procedures. They are applied in certain circumstances, e.g. water samples from the pulp and paper industry are analyzed for approximately 250 organic compounds, some of which are unique to this industry.

3. Analysis of organic compounds not identified as 'target compounds' or included in the 'Treated Water Survey' or the pulp and paper industry list. Detailed information has been and continues to be collected by the Pollution Control Division on the the identification and quantification of many additional organic compounds which, at present, do not constitute an environmental threat. This information may prove valuable in the future.

The Pollution Control Division has well equipped laboratories and experienced senior scientific personnel and technical staff to conduct the analyses of organic compounds in aqueous samples.

Two low resolution mass spectrometers are used extensively in analytical procedures. The bench-top instrument (HP model 5995) is a dedicated instrument; it is used exclusively for the analysis of purgable volatile compounds. The more versatile and sophisticated instrument (HP model 5987) is used extensively for the identification of extractable organic acids, bases and neutral compounds. A new instrument (HP model 5970) will soon be available. It will be used extensively in combination with GC as a mass selective detector.

There are at least 10 gas chromatographs in regular used in the Division. Some have single detectors; some have two detectors, and some have the capability of providing simultaneous detection of separated organic compounds by three different detectors. This versatility greatly improves the analyst's ability to identify and quantify. The gas chromatographs are employed in the routine analysis of common pollutants, in research problems directed towards the improvement of analytical procedures, and prior to GC/MS analyses. All gas chromatographic analyses are conducted on capillary GC columns, thus ensuring maximal separation of extracted organic compounds.

The Pollution Control Division also uses HPLC techniques in some analytical procedures, and is well equipped with liquid chromatographs for this purpose.

Conclusion: This is an excellent facility, comparable in expertise (but not in size) to the analytical services offered by the Water Resources Branch in Toronto. The procedures used to isolate, separate, identify and quantify trace organics in water and other environmental samples are 'state of the art'. The personnel involved in setting up the analytical procedures are experienced and knowledgable, and aware of the current literature on analytical techniques. I was impressed by their expertise.

SAMPLING REQUIREMENTS: EACH SAMPLE MUST BE SUBMITTED IN AN APPROPRIATE CONTAINER, PROVIDED BY THE CHEMISTRY WING OF ALBERTA ENVIRONMENTAL CENTRE (TELEPHONE - VEGREVILLE 632-6761 EXT. 279).

WATER .... EACH SCAN REQUIRES A SEPARATE ONE LITRE AMBER BOTTLE FILLED WITH NO HEAD SPACE. PURGEABLE AND VOLATILE SCANS REQUIRE A ONE LITRE AMBER BOTTLE MARKED "VOLATILE".

SOIL & SEDIMENT.... EACH SCAN REQUIRES A NEAR FULL 500 ML AMBER OINTMENT JAR.

TRACE ORGANIC CONTAMINANT SCANS

I. EXTRACTABLE PRIORITY POLLUTANTS

a. Acids

Benzoic acid
4-Chloro-*m*-cresol
2-Chlorophenol
2,4-Dichlorophenol
2,4-Dimethylphenol
4,6-Dinitro-*o*-cresol
2,4-Dinitrophenol
Linoleic acid
Linolenic acid
Hexadecanoic acid
2-Nitrophenol
4-Nitrophenol
Oleic acid
Pentachlorophenol
Phenol
2,4,5-Trichlorophenol
2,4,6-Trichlorophenol

b. Polynuclear Aromatics

Acenaphthylene
Anthracene
Benz(a)anthracene
Benzo(k)fluoranthene
Benzo(ghi)perylene
Benzo(a)pyrene
Chrysene
Dibenz(ah)anthracene
Fluoranthene
Fluorene
Indeno(1,2,3-cd)pyrene
Naphthalene
Perylene
Phenanthrene
Pyrene

c. Chloroorganics

2-Chloronaphthalene
Hexachlorobenzene
Hexachlorobutadiene
Hexachlorocyclopentadiene
Hexachloroethane
Pentachlorobenzene
1,2,3,5-Tetrachlorobenzene
1,2,4,5-Tetrachlorobenzene
1,2,4-Trichlorobenzene

d. Nitroaromatics

Benzidine
3,3'-Dichlorobenzidine
2,4-Dinitrotoluene
2,6-Dinitrotoluene
1,2-Diphenylhydrazine
Nitrobenzene
N-Nitrosodimethylamine
N-Nitrosodiphenylamine
N-Nitrosodi-*n*-propylamine

e. Halo Ethers

4-Bromophenyl phenyl ether
Bis(2-chloroethoxy)methane
Bis(2-chloroethyl)ether
Bis(2-chloroisopropyl)ether
2-Chloroethyl vinyl ether
4-Chlorophenyl phenyl ether

f. Organophosphates

p-*t*Butylphenyl diphenyl phosphate
Ethylhexyl diphenyl phosphate
Tributyl phosphate
Tri(2-chloroethyl)phosphate
Tri-*o*-cresyl phosphate
Tri-*m*-cresyl phosphate
Tri-*p*-cresyl phosphate
Triphenyl phosphate
Tri(2,3-dichloropropyl)phosphate
Tri(2,4-xylenyl)phosphate

g. Phthalate Esters

Butylbenzyl phthalate
Di-*n*-butylphthalate
Diethylphthalate
Dimethylphthalate
Di-*n*-octylphthalate
Bis(2-ethylhexyl)phthalate

h. Miscellaneous

Benzothiazole
Caffeine
Isophorone

i. Others

II. PURGEABLE PRIORITY POLLUTANTS

Benzene
Bromodichloromethane
Bromoform
Carbon tetrachloride
Chlorobenzene
Chloroform
Dibromochloromethane
1,2-Dichlorobenzene
1,3-Dichlorobenzene
1,4-Dichlorobenzene
1,1-Dichloroethane
1,1-Dichloroethylene
trans-1,2-Dichloroethylene
trans-1,3-Dichloropropylene
Ethyl benzene
Methylene chloride
1,1,2,2-Tetrachloroethane
Tetrachloroethylene
Toluene
Trichloroethylene
Trichlorofluoromethane
o-Xylene
m-Xylene
p-Xylene

III. VOLATILE SCAN

IV. PCB

V. HYDROCARBON SCAN

PESTICIDE SCANS

VI. ORGANOCHLORINE

Aldrin
α-Chlordane
γ-Chlordane
Captan
Dacthal®
(Chlorthal methyl)
o,p' - DDD
p,p' - DDD
o,p' - DDE
p,p' - DDE
o,p' - DDT
p,p' - DDT
Diallate (Avadex®)
Dieldrin
Dursban®
(Lorsban®, Chlorpyrifos)
α-Endosulfan
β-Endosulfan
Endrin
Heptachlor
Heptachlor epoxide
Hexachlorobenzene (HBC)
α-Hexachlorocyclohexane
β- "
γ- " (Lindane)
δ- "
Kelthane® (Dicofol)
Methoxychlor
Mirex
Perthane®
Ramrod® (Propachlor)
Triallate (Avadex BW®)

VII. ORGANOPHOSPHORUS/ ORGANONITROGEN

a. Organophosphorus

Aspon®
Crufomate (Ruelene®)
Dasanit® (Fensulfothion)
DDVP
(Dichlorovos, Vapona®)
DEF®
Demeton (Systox®)
Dimethoate (Cygon®)
Di-Syston® (Disulfoton)
Ethion
Fenthion®
Fenitrothion
Fonofos (Dyfonate®)
Guthion®
Malathion®
Methyl Parathion
Methyl Trithion®
Mevinphos® (Phosdrin®)
Mocap®
Paraoxon®
Parathion
Phorate (Thimet®)
Ronnel (Fenchlorphos®)
Tetrachlorvinphos (Gardona®)

b. Organonitrogen

Atrazine
Aminocarb (Matacil®)
Bromacil (Isocil)
Cobex® (Dinitramine®)
Cyanazine (Bladex®)
Diazinon
Dinoseb
Metribuzin (Sencor®)
Prometryne
Propanil
Propazine
Simazine
Terbacil
Trifluralin (Treflan®)

VIII. SELECTED ORGANONITROGEN

Barban
Carbaryl
Carbofuran (Furadan®)
Difenzoquat (Avenge®)
Dimilin®
Diquat (Reglone®)
Diuron
Linuron®
Monuron
Paraquat
Propoxur (Baygon®)
Pyrazon
Rotenone

IX. HERBICIDE ACIDS

2,4-D
Dicamba (Banvel®)
Diclofop-methyl
(Hoe-Grass®)
MCPA
2,4,5-T
Picloram (Tordon®)

X. OTHERS

Alberta
ENVIRONMENTAL CENTRE

BAG 4000, VEGREVILLE, ALBERTA T0B 4L0

Chemical Analysis Request

Methods Development and Research Services Section

(IMPORTANT: see reverse side for sample requirements)

Results to ....
(name)

....
(address) **(telephone)**

Sample Type	**Ground Water**	**Soil**	**Plant** ....
	Surface Water	**Sediment**	**Biota** ....
	Industrial Effluent	**Sludge**	**Other** ....

Program/Project .... **Is this for human consumption?** ....

Sampling Location ....

....

Date Sampled .... **Sampled by** ....

Analysis Required (please specify) ....

....

....

....

(To be completed by Methods Development and Research Services Section)

Date Received .... **Received by** .... **Sample No** ....

Table I. Purged and trapped organic volatiles analyzed by GC.

SAMPLE DESCRIPTION											
PAGE # BOOK #											
SUBMISSION #											
LIS # OF SAMPLE											
1,1-Dichloroethylene											
Methylene Chloride											
Trans-1,2-Dichloroethylene											
1,1-Dichloroethane											
Chloroform											
1,1,1-Trichloroethane											
1,2-Dichloroethane											
Carbontetrachloride											
Benzene											
1,2-Dichloropropane											
Trichloroethylene											
Dichlorobromomethane											
Toluene											
1,1,2-Trichloroethane											
Chlorodibromomethane											
Tetrachloroethylene											
Chlorobenzene											
Ethylbenzene											
m- and p-xylene (RMP)											
Bromoform											
o-xylene											
1,1,2,2-Tetrachloroethane											
1,4-Dichlorobenzene											
1,3-Dichlorobenzene											
1,2-Dichlorobenzene											
TOTAL TRIHALOMETHANES											
NOTES + REMARKS											

Table II. Organic compounds analyzed by GC

Drinking Water Unit Parameter Analyses List - Nov7/85

Chlorinated Organic Compounds (OWOC)

Compound	DL(ppt)	Compound	DL(ppt)
Hexachlorobutadiene	1	A-BHC Hexachlorocyclohexane	1
Hexachloroethane	1	B-BHC Hexachlorocyclohexane	1
135-Trichlorobenzene	5	G-BHC Hexachlorocyclohexane	1
124-Trichlorobenzene	5	A-Chlordane	2
123-Trichlorobenzene	5	G-Chlordane	2
1235-Tetrachlorobenzene	1	Oxychlordane	2
1245-Tetrachlorobenzene	1	op-DDT	5
1234-Tetrachlorobenzene	1	pp-DDT	5
Pentachlorobenzene	1	pp-DDD	5
Hexachlorobenzene	1	Methoxychlor	5
245-Trichlorotoluene	5	Heptachlor epoxide	1
236-Trichlorotoluene	5	Endosulfan I	2
26a-Trichlorotoluene	5	Endosulfan II	4
Octachlorostyrene	1	Endosulfan sulphate	4
Total PCBs	20	Dieldrin	2
Heptachlor	1	Endrin	4
Aldrin	1	Mirex	5
pp-DDE	5	Toxaphene (!MP)	200

Chlorinated Acid Herbicides (OWCP)

Chlorophenoxy Compound	DL(ppt)	Chlorobenzoic Compound	DL(ppt)
Dicamba	100	246-Trichlorophenol	50
24D	100	245-Trichlorophenol	50
24DB	200	234-Trichlorophenol	100
24DP	100	2356-Tetrachlorophenol	50
245T	50	2345-Tetrachlorophenol	50
Silvex	50	Pentachlorophenol	50
Picloram	100		

Organophosphate and Triazine Compounds (PWAOP,PWACP/PA)

Organophosphate Pesticide	DL(ppt)	Triazine Herbicide	DL(ppt)
Dichlorvos	20	Propazine	50
Phorate (Thimet)	20	Atrazine	50
Mevinphos	20	Simazine	50
Diazinon	20	Sencor	100
Ronnel	20	Bladex	100
Parathion	20	Prometone	50
Methyl Parathion	20	Ametryne	50
Malathion	20	Prometryne	50
Ethion	20	Atratone	50
Methyl Trithion	200		
Chlorpyriphos (Dursban)	20		
Reldan	20		
Azinphos Methyl (Guthion)	2000		

Table II. contd.

Carbamate and Phenyl Urea Compounds (PWACAR,PWAUN)

Carbamate Pesticide	DL(ppb)	Phenyl Urea Compound	DL(ppb)
Captan	2.0	Metoxuron	2.0
Carbofuran	2.0	Monuron	2.0
Propoxur	2.0	Chlortoluron	2.0
Carbaryl	2.0	Fluometuron	2.0
IPC	2.0	Diuron	2.0
Aminocarb (!NP)	2.0	Difenoxuron	2.0
CIPC	2.0	Monolinuron	2.0
Eptam	2.0	Linuron	2.0
Benomyl (!NP)	2.0	Patoran	2.0
Bux (!NP)	2.0	Metobromuron	2.0
Barban	2.0	Chlorbromuron	2.0
Butylate (sutan)	2.0	Siduron	2.0
Diallate	2.0	Neburon	2.0

Alachlor (LASS)

Compound	DL(ppb)
Alachlor	1.0

DL = Detection Limit

Drinking Water Test Codes

Test Group	LIS Code
OC / CB / PCBs / Toxaphene	DWOC
PCBs only	P1PCBT
Chlorophenols / Phenoxyacids	DWCP
Organo Phosphorus Compounds	PEOP
Triazines	PETRI
Carbamates	PECAR
Phenyl Ureas	PEUN
Alachlor (Lasso)	POLASS

Table II. contd.

LIST OF COMPOUNDS TESTED FOR :

P.A.H.

1) PHENANTHRENE
2) ANTHRACENE
3) FLUORANTHENE
4) PYRENE
5) BENZO (A) ANTHRACENE
6) CHRYSENE
7) DIMETHYL BENZO (A) ANTHRACENE
8) BENZO (E) PYRENE
9) BENZO (J) FLUORANTHENE
10) BENZO (B) FLUORANTHENE
11) PERYLENE
12) BENZO (K) FLUORANTHENE
13) BENZO (A) PYRENE
14) BENZO (G H I) PERYLENE
15) DIBENZO (A H) ANTHRACENE
16) INDENO (1,2,3 - C, D) PYRENE
17) BENZO (B) CHRYSENE
18) ANTHANTHRENE
19) CORONENE

PHENOLS
1) HOMOVANILIC ACID
2) PHENOL
3) GUAICOL
4) VANILLINE
5) ACETOVANILLONE
6) ACETOSYRINGON

Table III. Nonvolatile extractable compounds analyzed by GC

LIS Submission #

LIS Sample #

PP Reference #

Sample Location:

Date Analysed:

Base/Neutrals

	DETECTION LIMITS IN PPB	
1. N-Nitroso-Dimethylamine	4	
2. N-Nitroso-Di-n-Propylamine	1	
3. Nitrobenzene	2	
4. ~~Hexachloroethane~~	~~2~~	
5. Bis-(2-chloroethoxy)methane	2	
6. ~~1,2,4 Trichlorobenzene~~	1	
7. Naphthalene	1	
8. p-Chloroaniline		
9. ~~Butoxyethoxyethanol~~	50	
10. Benzothiazole	10	
11. ~~Hexachlorobutadiene~~	2	
12. Hexachlorocyclopentadiene	15	
13. 2-Chloronaphthalene	1	
14. 2,6-Dinitrotoluene	1	
15. ~~Acenaphthylene~~	1	
16. Di-methyl Phthalate	2	
17. ~~Acenaphthene~~	1	
18. 2,4-Dinitrotoluene	2	
19. Di-t-butyl Methyl Phenol	4	
20. ~~Fluorene~~	1	
21. Di Ethyl Phthalate	1	
22. 1,2-Diphenyl Hydrazine	1	
23. 4-Bromophenyl Phenyl Ether	1	
24. ~~Hexachlorobenzene~~	2	
25. ~~Phenanthrene~~	1	
26. ~~Anthracene~~	1	
27. ~~Caffeine~~		
28. Di n-Butyl Phthalate	1	
29. ~~Fluoranthene~~	1	
30. ~~Octachlorostyrene~~	50	
31. ~~Pyrene~~	1	
32. Butyl Benzyl Phthalate	1	
33. ~~Benzo(a)Anthracene~~	1	
34. ~~Chrysene~~	1	
35. Bis-(2-Ethylhexyl) Phthalate	1	
36. ~~Benzo(b)Fluoranthene~~	2	
37. Di-n-Octyl Phthalate	1	
38. ~~Benzo(a)Pyrene~~	5	
39. ~~Benzo(k)Fluoranthene~~	20	
40. ~~Dibenzo(a,h)Anthracene~~		
41. ~~Cholesterol~~		
42. ~~Benzo(g,h,i)Perylene~~		
43. 4-Chlorophenyl Phenyl Ether	1	

Acids

	Detection Limits in PPB	
1. Phenol	4	
2. 2-Chloro Phenol	4	
3. 2-Nitrophenol	10	
4. 2,4-Dimethyl Phenol	2	
5. 2,4-Dichlorophenol	4	
6. p-Chloro-m-Cresol	2	
7. 2,4,6-Trichlorophenol	4	
8. 2,4-Dinitrophenol	70	
9. 4-Nitrophenol	10	
10. 4,6-Dinitro-o-Cresol	80	
11. Pentachlorophenol	5	
12. ~~Hexadecanoic Acid~~	~~75~~	

Ministry
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ntario

Table IV. Volatile organic compounds analyzed by GC/MS.

Components Found & Their Concentration in ug/L

Date Sampled: ____________________

Date Analyzed: ____________________ Sample ____________________

Compound					
Methylene Chloride					
1,1-Dichloroethylene					
I.S Bromochloromethane					
1,1-Dichloroethane					
Chloroform					
1,2-Dichloroethane					
1,1,1-Trichloroethane					
Carbontetrachloride					
Dichlorobromomethane					
1,2-Dichloropropane					
Benzene					
Trichloroethylene					
1,1,2-Trichloroethane					
Chlorodibromomethane					
1,2-Dibromoethane					
Bromoform					
I.S 1,3-Dichlorobutane					
Tetrachloroethylene					
Toluene					
1,1,2,2-Tetrachloroethane					
Chlorobenzene					
Ethylbenzene					
m-Xylene					
o- or p-Xylene					
1,2-Dichlorobenzene					

OTES

ND	- not detected	- less 0.05 ug/litre
ND†	- not detected	- less than 5 ug/litre
tr	- trace	- less than 0.1 ug/litre
*	- approximate	- response of standard not determined

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Table V. Lists of DWSP Compounds.

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
1 ALKALINITY 1 (LAB) mg/L	0.2 mg/L	
2 ALUMINUM 2 (MET) mg/L	0.003 mg/L	
3 ARSENIC 3 (MET) mg/L	0.001 mg/L	0.05 mg/L
4 BARIUM 4 (MET) mg/L	0.001 mg/L	1 mg/L
5 BORON 5 (MET) mg/L	0.02 mg/L	5 mg/L
6 BERYLLIUM 6 (MET) mg/L	0.001 mg/L	
7 BENZENE 7 (VOL) ug/L	1 ug/L	10 ug/L h
8 TOLUENE 8 (VOL) ug/L	1 ug/L	14300 ug/L e
9 ETHYLBENZENE 9 (VOL) ug/L	1 ug/L	1400 ug/L e
10 P-XYLENE 10 (VOL) ug/L	1 ug/L	620 ug/L e
11 M-XYLENE 11 (VOL) ug/L	1 ug/L	620 ug/L c

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
12 O-XYLENE 12 (VOL) ug/L	1 ug/L	620 ug/L c
13 CALCIUM 13 (LAB) mg/L	0.1 mg/L	
14 CYANIDE 14 (MET) mg/L	0.001 mg/L	0.2 mg/L
15 CADMIUM 15 (MET) mg/L	0.0003 mg/L	0.005 mg/L
16 CHLORIDE 16 (LAB) mg/L	0.2 mg/L	250 mg/L
17 COLOUR TCU 17 (LAB)	0.5 TCU	5 TCU
18 CONDUCTIVITY 18 (LAB) umho/cm	0.01 UMHO/CM	
19 COBALT 19 (MET) mg/L	0.001 mg/L	
20 CHROMIUM 20 (MET) mg/L	0.001 mg/L	0.05 mg/L
21 COPPER 21 (MET) mg/L	0.001 mg/L	1 mg/L
22 F. COLIFORM MF 22 (BAC) count/100mL	0	0/0.1L mL

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
23 IRON 23 (MET) mg/L	0.002 mg/L	0.3 mg/L
24 FLUORIDE 24 (LAB) mg/L	0.01 mg/L	2.4 mg/L
25 FIELD CHLORINE (COMBINED) 25 (FLD)		
26 FIELD CHLORINE (FREE) 26 (FLD)		
27 FIELD CHLORINE (TOTAL) 27 (FLD)		
28 FIELD PH 28 (FLD)		
29 FIELD TEMPERATURE 29 (FLD)		
30 FIELD TURBIDITY 30 (FLD)		1 FTU
31 HARDNESS 31 (LAB) mg/L	0.5 mg/L	
32 STANDARD PLATE COUNT MF 32 (BAC) count/mL	0	500 organisms per mL
33 MERCURY 33 (MET) ug/L	0.01 ug/L	1 ug/L

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
34 MAGNESIUM 34 (LAB) mg/L	0.05 mg/L	
35 MANGANESE 35 (MET) mg/L	0.001 mg/L	0.05 mg/L
36 MOLYBDENUM 36 (MET) mg/L	0.001 mg/L	0.25 mg/L a
37 SODIUM 37 (LAB) mg/L	0.1 mg/L	
38 NICKEL 38 (MET) mg/L	0.002 mg/L	
39 AMMONIUM TOTAL 39 (LAB) mg/L	0.05 mg/L	
40 NITRITE 40 (LAB) mg/L	0.005 mg/L	1 mg/L as N
41 NITRATE 41 (LAB) mg/L	0.05 mg/L	10 mg/L as N
42 NITROGEN TOTAL KJELDAHL 42 (LAB) mg/L	0.1 mg/L	0.15 mg/L *
43 PRESENCE/ABSENCE 43 (BAC)	0	Absent
44 LEAD mg/L 44 (MET)	0.003 mg/L	0.05 mg/L

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
45 PH 45 (LAB)		
46 PHOSPHORUS FILTERED REACTIVE 46 (LAB) mg/L	0.01 mg/L	
47 PHOSPHORUS TOTAL 47 (LAB) mg/L	0.01 mg/L	
48 ALDRIN 48 (PST) ng/L	1 ng/L	700 ng/L **
49 ALPHA BHC 49 (PST) ng/L	1 ng/L	700 ng/L c
50 BETA BHC 50 (PST) ng/L	1 ng/L	300 ng/L c
51 LINDANE 51 (PST) ng/L	1 ng/L	4000 ng/L
52 ALPHA CHLORDANE 52 (PST) ng/L	2 ng/L	700 ng/L ***
53 GAMMA CHLORDANE 53 (PST) ng/L	2 ng/L	700 ng/L ***
54 DIELDRIN 54 (PST) ng/L	2 ng/L	700 ng/L **
55 METHOXYCHLOR 55 (PST) ng/L	5 ng/L	100000 ng/L

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
56 ENDRIN 56 (PST) ng/L	4 ng/L	200 ng/L
57 THIODAN SULPHATE 57 (PST) ng/L	4 ng/L	
58 THIODAN I 58 (PST) ng/L	2 ng/L	74000 ng/L ea
59 THIODAN II 59 (PST) ng/L	4 ng/L	74000 ng/L ea
60 METHYLPARATHION 60 (SPC)	50 ng/L	7000 ng/L
61 PARATHION 61 (SPC)	50 ng/L	35000 ng/L
62 HEPTACHLOR EPOXIDE 62 (PST) ng/L	1 ng/L	3000 *** ng/L
63 HEPTACHLOR 63 (PST) ng/L	1 ng/L	3000 ng/L ***
64 MIREX 64 (PST) ng/L	5 ng/L	
65 OXYCHLORDANE 65 (PST) ng/L	2 ng/L	
66 O,P,-DDT 66 (PST) ng/L	5 ng/L	30000 ng/L d

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
67 PCB 67 (PST) ng/L	20 ng/L	3000 ng/L t
68 P,P-DDD 68 (PST) ng/L	5 ng/L	d
69 P,P-DDE 69 (PST) ng/L	1 ng/L	d
70 P,P-DDT 70 (PST) ng/L	5 ng/L	d
71 AMETRINE 71 (SPC) ng/L	50 ng/L	
72 ATRAZINE 72 (SPC) ng/L	50 ng/L	
73 DIAZINON 73 (SPC) ng/L	50 ng/L	14000 ng/L
74 BLADEX 74 (SPC) ng/L	100 ng/L	
75 PROMETONE 75 (SPC) ng/L	50 ng/L	
76 PROPAZINE 76 (SPC) ng/L	50 ng/L	
77 PROMETRYNE 77 (SPC) ng/L	50 ng/L	

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
78 SENCOR 78 (SPC) ng/L	100 ng/L	
79 SIMAZINE 79 (SPC) ng/L	50 ng/L	
80 DICAMBA 80 (SPC) ng/L	100 ng/L	
81 PICLORAM 81 (SPC) ng/L	100 ng/L	
82 SILVEX 82 (SPC) ng/L	50 ng/L	10000 ng/L
83 2,4-D 83 (SPC) ng/L	100 ng/L	100000 ng/L
84 2,4-D BUTYRIC ACID 84 (SPC) ng/L	200 ng/L	
85 2,4-D PROPIONIC ACID 85 (SPC) ng/L	100 ng/L	
86 2,4,5-T 86 (SPC) ng/L	50 ng/L	
87 TOTAL SOLIDS 87 (LAB) mg/L	1 mg/L	
88 SELENIUM 88 (MET) mg/L	0.001 mg/L	0.01 mg/L

Table V. (contd) Lists of DWSP Compounds

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
89 STRONTIUM 89 (MET) mg/L	0.001 mg/L	
90 TOTAL COLIFORM MF 90 (BAC) count/100mL	0	ODWO Bacti
91 TOTAL COLIFORM BACKGROUND MF 91 (BAC) count/100mL	0	ODWO Bacti
92 TURBIDITY 92 (LAB) FTU	0.01 FTU	1 FTU
93 URANIUM 93 (MET) mg/L	0.002 mg/L	.02 mg/L t
94 VANADIUM 94 (MET) mg/L	0.001 mg/L	
95 HEXACHLOROBUTADIENE 95 (CHA) ng/L	1 ng/L	4500 ng/L e
96 1,1-DICHLOROETHYLENE 96 (VOL) ug/L	1 ug/L	.3 ug/L h
97 METHYLENE CHLORIDE 97 (VOL) ug/L	5 ug/L	40 ug/L c
98 T,1,2-DICHLOROETHYLENE 98 (VOL) ug/L	1 ug/L	
99 1,1-DICHLOROETHANE 99 (VOL) ug/L	1 ug/L	

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
100 CHLOROFORM 100 (VOL) ug/L	1 ug/L	350 ug/L ++
101 DICHLOROMETHANE 101 (VOL) ug/L	5 ug/L	40 ug/L c
102 1,1,1-TRICHLOROETHANE 102 (VOL) ug/L	1 ug/L	
103 DICHLOROETHANE 103 (VOL) ug/L	1 ug/L	
104 1,2-DICHLOROETHANE 104 (VOL) ug/L	1 ug/L	10 ug/L h
105 CARBON TETRACHLORIDE 105 (VOL) ug/L	1 ug/L	3 ug/L h
106 1,2 DICHLOROPROPANE 106 (VOL) ug/L	1 ug/L	6 ug/L ep
107 TRICHLOROETHYLENE 107 (VOL) ug/L	1 ug/L	30 ug/L h
108 DICHLOROBROMOMETHANE 108 (VOL) ug/L	1 ug/L	350 ug/L ++
109 1,1,2-TRICHLOROETHANE 109 (VOL) ug/L	1 ug/L	6 ug/L e
110 CHLORODIBROMOMETHANE 110 (VOL) ug/L	1 ug/L	350 ug/L ++

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
111 TETRACHLOROETHYLENE 111 (VOL) ug/L	1 ug/L	10 ug/L h
112 BROMOFORM 112 (VOL) ug/L	1 ug/L	350 ug/L ++
113 1,1,2,2-TETRACHLOROETHANE 113 (VOL) ug/L	1 ug/L	1.7 ug/L e
114 HEXACHLOROBENZENE 114 (PST) ng/L	1 ng/L	10 ng/L h
115 HEXACHLOROETHANE 115 (CHA) ng/L	1 ng/L	19000 ng/L e
116 OCTACHLOROSTYRENE 116 (CHA) ng/L	1 ng/L	
117 PENTACHLOROBENZENE 117 (CHA) ng/L	1 ng/L	74000 ng/L e
118 TOTAL TRIHALOMETHANES 118 (VOL) ug/L	3 ug/L	350 ug/L ++
119 2,3,6-TRICHLOROTOLUENE 119 (CHA) ng/L	5 ng/L	
120 2,4,5-TRICHLOROTOLUENE 120 (CHA) ng/L	5 ng/L	10000 ng/L g
121 2,6,A-TRICHLOROTOLUENE 121 (CHA) ng/L	5 ng/L	

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
122 CHLOROBENZENE 122 (VOL) ug/L	1 ng/L	100-300 ng/L h*
123 1,4-DICHLOROBENZENE 123 (VOL) ug/L	1 ug/L	400 ug/L e
124 1,3-DICHLOROBENZENE 124 (VOL) ug/L	1 ug/L	400 ug/L e
125 1,2-DICHLOROBENZENE 125 (VOL) ug/L	1 ug/L	400 ug/L e
126 TRIFLUOROCHLOROTOLUENE 126 (CHA) ug/L	1 ug/L	
127 1,2,3-TRICHLOROBENZENE 127 (CHA) ng/L	5 ng/L	10000 ng/L y
128 1,2,3,4-TETRACHLOROBENZENE 128 (CHA) ng/L	1 ng/L	38000 ng/L e
129 1,2,3,5-TETRACHLOROBENZENE 129 (CHA) ng/L	1 ng/L	
130 1,2,4-TRICHLOROBENZENE 130 (CHA) ng/L	5 ng/L	15000 ng/L y
131 1,2,4,5-TETRACHLOROBENZENE 131 (CHA) ng/L	1 ng/L	38000 ng/L e
132 1,3,5-TRICHLOROBENZENE 132 (CHA) ng/L	5 ng/L	10000 ng/L y

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
144 PENTACHLOROBUTADIENE 144 (MS) ug/L	0.1 ug/L	
145 N-DICHLOROMETHYLENE- 145 PENTACHLOROANALINE 145 (MS) ug/L	0.1 ug/L	
146 FLUORANTHENE 146 (MS) ug/L	0.1 ug/L	
147 NAPHTHALENE 147 (MS) ug/L	0.1 ug/L	
148 METHYL PHENANTHRENE 148 (MS) ug/L	0.1 ug/L	
149 PYRENE 149 (MS) ug/L	0.1 ug/L	
150 DIPHENYL ETHER 150 (MS) ug/L	0.1 ug/L	
151 DI-N-BUTYL PHTHALATE 151 (MS) ug/L	0.1 ug/L	
152 CL BIPHENYL 152 (MS) ug/L	0.1 ug/L	
153 ATRAZINE 153 (MS) ug/L	0.1 ug/L	

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
154 CARBON TETRACHLORIDE 154 (MS) ug/L	0.1 ug/L	
155 DIBROMOCHLOROMETHANE 155 (MS) ug/L	0.1 ug/L	
156 TETRACHLOROETHYLENE 156 (MS) ug/L	0.1 ug/L	
157 BIPHENYL 157 (MS) ug/L	0.1 ug/L	
158 BROMOFORM 158 (MS) ug/L	0.1 ug/L	

PARAMETERS	DETECTION LIMIT	DRINKING WATER OBJ/ GUIDELINE[1]
133 PENTACHLOROPHENOL 133 (CHP) ng/L	50 ng/L	10000 ng/L h
134 2,3,4-TRICHLOROPHENOL 134 (CHP) ng/L	100 ng/L	
135 2,3,4,5-TETRACHLOROPHENOL 135 (CHP) ng/L	50 ng/L	
136 2,3,5,6-TETRACHLOROPHENOL 136 (CHP) ng/L	50 ng/L	
137 2,4,5-TRICHLOROPHENOL 137 (CHP) ng/L	50 ng/L	
138 2,4,6-TRICHLOROPHENOL 138 (CHP) ng/L	50 ng/L	10000 ng/L h
139 ZINC 139 (MET) mg/L	0.001 mg/L	5 mg/L h
140 PENTACHLOROPROPANE 140 (MS) ug/L	0.1 ug/L	
141 PENTACHLOROPROPENE 141 (MS) ug/L	0.1 ug/L	
142 HEXACHLOROPROPENE 142 (MS) ug/L	0.1 ug/L	
143 TETRACHLOROBUTANE 143 (MS) ug/L	0.1 ug/L	

LAB - Chemistry (LAB)
FLD - Chemistry (FIELD)
BAC - Bacteriological
MS - Mass Spec Ana.

MET - Metal
VOL - Volatiles
PST - PCB/OC Scan. Pesticides

CHA - Chloroaromatics
CHP - Chlorophenols
SPC - Specific Pesticides

EVALUATION

OF

CITY OF EDMONTON

WATER TREATING PLANTS

PREPARED BY
P.D. FOLEY ENGINEERING LTD.
May, 1986

EVALUATION OF CITY OF EDMONTON WATER TREATING PLANTS

The purpose of this report is to:

1. Review the Operating Licence as used in Alberta and compare to methods of control of Water Utilities used in Quebec and Ontario.

2. Compare the technology and practice used in the Edmonton water treatment plants with Canadian experience.

OPERATING LICENCE

The methods used in the three Provinces, Alberta, Quebec and Ontario for the control of the construction and operation of facilities for the treatment of potable water are very similar. All three have legislation in place which controls the construction of plants by permit with an approval issued on completion. The same is true for any modifications or additions made at a later date. The three Provinces do differ in the reporting arrangements for the quality and quantity of water produced and Alberta is the only Province with a Licence document. Quebec achieves the same result by putting very similar reporting requirements into a directive issued by the Environment Ministry under the enabling legislation and is the only Canadian Province to incorporate legally enforceable drinking water standards into legislation. As a result, both Alberta and Quebec require the submission of similar information to the Ministry on a monthly basis.

Ontario, because for most water works the Environment Ministry provides analytical services, has had no need for a similar reporting arrangement. Copies of all analysis are sent to the responsible Ministry staff when the originals go out to the utility. For those large utilities that do their own analysis, they supply similar information monthly and annually as is supplied in Alberta and Quebec. In addition, those small utilities, for whom the Province provides analytical services, must provide annually information on quantities of water produced.

In summary, it would appear that Alberta is already one of the leading Provinces in the development of systems to provide control of water works and protection for its citizens.

TECHNOLOGY AND PRACTICE

In general, the unit processes used in the various treatment trains at the Edmonton Water Treatment Plants are similar to those that will be seen in other Provinces. Rates and retention times do vary. Filter rates in Quebec are limited to 4 USGPM per sq. ft., in Ontario to 4.9 USGPM per sq. ft. and in the rebuilt plants 1 and 2 at Edmonton, when operating at 270 MLPD, rates are 3.2 USGPM per sq. ft. with all nine filters in service, 3.60 with eight, and 4.1 with seven. Ontario will allow higher rates, up to 6.1 with demonstrated influent and effluent quality. The effluent quality objectives and the sampling requirements are as outlined in the Federal publication "Guidelines for Canadian Drinking Water Quality", in all three jurisdictions. Overall, any differences between the three jurisdictions are minor.

During the inspection of the Edmonton plants in December, some components, eg. mixers, were missing but are to be installed during the present rebuilding program, except for Plant #3. Others such as the horizontal flow softening units in Plants 1 and 2 were not operating satisfactorily. This would appear to be due to inadequacies of design brought on by the series/parallel operation of the clarifiers and the resulting requirement that the softening be done in a unit designed for clarification rather than softening. Finally, a river bank intake by its very nature is ideally located to pick up any poor quality water discharged from the land and would be the worst possible location from which to take drinking water, even in winter it is at risk.

The provision of the new 75 and 100 HP mixers should eliminate the main mixing problems at Plants 1 and 2. However, I am not in favour of the addition of multiple chemicals at a single mixer, as is practiced at Edmonton. If possible, I would use the mixer for alum and if necessary a disinfectant. I would never include alkalies such as ammonia or lime at the same feed point. The carbon should be fed as far up stream as possible to provide available contact time before being enmeshed in the alum floc.

FLOCCULATION

The new tapered flocculation units should provide best results obtainable. The maximum flow through Plant #1 of 100 MLPD allows only a 23 minute theoretical floc time which I would consider a minimum time, assuming a normal short circuiting value. At the normal high flow of 75 MLPD, the retention increases to 30 minutes, a more normal flocculation time. Slightly lower times based on different qualtities would apply to Plant #2.

SEDIMENTATION

The maximum rise rate through the tubes on Plants 1 and 2 is 3.2 gpm/sq. ft. This rate is low for softening but high for clarification. An improved quality effluent could be achieved with the use of weighters such as clay or lime and with the use of a coagulant aid such as activated silica. At the normal high flow of 75 MLPD the rise rate in Plant #1 is 2.4 gpm/sq. ft., a level that I consider the maximum without the use of aides. Better than normal solids removal would probably assist in reducing taste and odours, protecting against giardia cysts, reduce water borne contaminants and by reducing organics could improve the subsequent softening phase.

SOFTENING

Since Plants 1 and 2 are designed for parallel/series operation, the two halves of the Plant are identical. Unfortunately, the treatment times required for optimum clarification differ from those required for softening. Mixing times for softening should run 5 to 10 minutes rather than 30 seconds in order to provide for the slow disolving of the calcium hydrate. The flocculation time needs to be increased also from the normal 30 minutes to more in the order of 40-60 minutes. The additional time allows for the rather slow precipitation of the new calcium carbonate on the old floc particles and for them to grow to a settleable size. The longer flocculation times will be achieved when operating the Plant in the parallel mode. At a flow of 75 MLPD, the flocculation time would increase to 60 minutes and at 100 MLPD to 40 minutes. There would still appear to be a problem with inadequate mixing even with the extended flocculation times. When operating in the series mode additional mixing time might be attained by the installation of mixers in the recarbonation channel and moving the chemical dosing point further up stream to provide some additional time. Unfortunately, I have no information on the size of the channel and the practicality of the suggestion.

None of the above times makes any allowance for short circuiting in the flocculators. It always takes place and plant personnel should determine the degree. If it is excessive steps should be taken to correct the problem as the theoretical flocculation times, V/Q are already low.

The use of tube settlers generally eliminates any problems associated with short circuiting during sedimentation. They have shown excellent capacity to smooth out over flow quality and one of their side benefits is the coagulation effect and for that matter the softening reaction time provided through and under the tubes. I have made no allowance for these reactions in the foregoing discussion on softening as I have no direct experience with horizontal flow softening systems to which I could relate.

FILTERATION

The average effluent turbidity was 0.5 NTU in 1984 and is slightly higher than the 0.3 NTU value considered, at least in Ontario, to be normal. To provide for removal of giardia cysts, it would be wise to try to be as near 0.1 NTU as possible. In addition, the lower turbidity could increase organic removal and reduce taste and odour problems as well as provide for removal of other organics of potential health significance. An indication of the methods required and the benefits can be determined in jar tests. Such tests should form an integral part of a program to resolve the taste and odour difficulties being experienced.

The effluent from #6 filters should be checked and compared to the other units. The sand probably has a greater effective size and a lower uniformity coefficient than the sand in the other filters. I have no idea what effect, if any, this might have on the quality of effluent or other operating parameters.

PLANTS 3 and 4

The comments on mixing and combined chemical feeds apply to these Plants.

Solids contact clarifiers are much more amenable to use as both clarifiers and softeners than are horizontal flow units. The carryover at Plant #4 from the softener seemed higher than necessary. We generally add a coagulant (activated silica works well) to hold the solids in the softener. The same is true when operating in the clarifier mode. As staff are already aware, activated silica in combination with the alum as the primary coagulant is most effective in minimizing clarifier discharge turbidity.

ONTARIO TREATMENT PLANTS

About 80% of Ontario's drinking water is drawn from the Great Lakes. Algae oriented taste and odour episods occur at times at virtually all Plants but in almost all cases can be controlled by an increase in the free chlorine residual. Some Plants are equipped for the emergency feeding of powdered activated carbon but the facilities are seldom if ever used.

Two Municipalities, Burlington and Oakville, located on Lake Ontario in the area of four petrolium refineries had taste problems caused by chlorination of phenols. Chlorine dioxide applied when required after filtration and prior to post chlorination resolved the problem. Subsequently, these Plants switched to continuous chlorine dioxide feed to catch the initial phenol. At about the same time the refineries installed proper discharge treatment systems and eliminated phenol and hydrocarbon discharges. In subsequent years a litany of taste and odour incidents variously described as medicinal, hydrocarbon or gasoline were reported and there seemed no solution to this taste problem. Taste and laboratory testing on raw water was negative. Some three years ago the Municipalities began to reduce the chlorine dioxide dosage with a similar reduction in complaints. No chlorine dioxide has been fed in the last two years and any taste and odour complaints have been mainly related to high algae counts identified by algae counting and to geosmin identified by chemical analysis. Problems were resolved by an increased free residual or by addition of PAC up to 10 PPM. The Great Lakes have relatively low trihalomethane levels. THMs at the two locations generally run 15 micrograms per litre in winter and up to 50 in the summer.

A second location at Belleville on the Bay of Quintee, off Lake Ontario, has a long history of adverse taste and odour at levels frequently exceeding 300 in summer and at 15 to 50 in the winter. The Ministry undertook extensive pilot plant tests a number of years ago. The basic treatment unit was a solids contact clarifier. Evaluation of potassium permanganate, free and combined chlorine, hydrogen peroxide, chlorine dioxide and powdered activated carbon fed to a small 30 minute retention tank ahead of the clarifier failed to reduce the threshold taste and odour to less than 16. Carbon columns would produce a taste free efluent. No ozonation was attempted but a later trial done at the Belleville Water Treatment Plant provided unsatisfactory results. A Treatment Plant was built further down the Bay based on the above, including carbon columns and provides taste free water.

The City of Brantford is located on the Grand River about 20 river miles below the Kitchener-Waterloo Region, a large industrial area. The City experienced almost continuous taste and odour problems in the fifties. These were resolved due to the work of Don Williams, the Plant Chemist.

The Plant has a nominal capacity of 12 MGPD and draws its water from the Grand via an old canal, and hence while the Plant is located in the City the supply is taken upstream of any urban runoff or sanitary overflows. To treat the water, twin uncovered treatment basins were constructed at the discharge from the canal, see attached Appendix A. Chlorine is dosed at 10 - 16 mg. per litre to provide 4.5 mg. per litre mainly free residual. The basin provides 60 min. retention at an average day and 35 min. at maximum. Powdered activated carbon is applied a mixer located on the barrier part way down the basin. Up to 50 mg. per litre of carbon are added. Carbon breakthrough occurs if this level is exceeded. Carbon retention time V/Q is 35 min. at average day flow and 22 minutes and maximum day flow.

This is one of the few Plants or possibly the only Plant in North America to use ammonia for dechlorination. Ammonia to reduce the residual and limit THM production is applied to the low lift pumps located at the basin discharge. Activated silica, which must be applied before the alum for best results, is also added at this point. Alum is added in a one minute mix tank and lime for PH correction is added to that discharge. Walking Beam flocculators provide 30 min. flocculation and conventional sedimentation basins follow. Filters are both dual media and tri media types. The microfloc tri media has been operated for extended periods at rates of up to 7 gpm. per sq. ft. for test purposes. The filter rate is 4.5 IGPM per sq. ft. at 21 MGPD, the maximum demand and 2.5 at 12 MGPD the nominal capacity.

They can feed either sulpher dioxide to dechlorinate or add more chlorine and ammonia at the discharge.

The converted installation eliminated all the taste and odour problems. Over the intervening years the upstream Municipalities and industries have improved their waste treatment systems and river water quality has drastically improved. Game fish are now caught in the river just below Brantford. As a result carbon feed is now only required during spring runoff and seldom at dosages exceeding 15 mg. per litre.

The City of Ottawa is located on the Ottawa River and like Edmonton has two treatment plants. One located on Lemieux Island in the middle of the river in the City centre. The other, Britannia, located on the river bank upstream of the City. Lemieux Island Plant has relatively few taste and odour incidents in spite of the fact that both storm water runoff and combined sewer overflows enter both sides of the river upstream from both treatment plants. Those that occur are algae related.

The new Plant, Britannia was completed in 1961, see Apendix B, and shortly after opening encountered taste and odour problems. The source could not be identified upstream and was finally found to be a back eddy from downstream. An extensiive raw water sampling program, (see AWWA Journal, Dec. 1981, "Raw Water Studies Determine Siting of New Intake", B.C. Hutcheon & D.W. Smith), led to the extension of the intake into an area of much improved water quality.

The Town of Dresden has a solids contact plant and is located on a river in an intensive agricultural area with a history of taste and odours that are related to rain fall and rural runoff. These are solved with potassium permanganate fed to the intake at a dose of 0.25 mg/L and results in a taste and odour tree product.

SUMMARY OF ONTARIO PLANTS

Most Ontario Plants operate based on similar parameters to those in Alberta. Taste and odour problems abound in summer but for the Great Lakes Plants are mainly algae related and easily resolved with excess chlorine. Since TOC normally run 3.5 mg. per litre, THMs do not present a problem through the excess chlorine. Maximum THM levels run at about 100 micrograms per litre. The Bay of Quintee is an exception and carbon columns are the only solution to the taste and odours encountered. The Dresden Plant described above is the only facility continuously feeding potassium permanganate.

We have experimented with permanganate elsewhere with reasonable success. It can be used successfully on Great Lakes waters to remove algae oriented taste and odours and is particularly effective on waters contaminated by rural runoff. Operator concern over potential colour discharges has always limited its use. While the development of a permanganate detector and controller has overcome that difficulty, it is still not commonly used where chlorine is as effective.

QUEBEC TREATMENT PLANTS

The writer also visited the Province of Quebec and spoken at length with Bernard Beauregard of the Montreal District of Environment Quebec. I also visited Plants in Laval (St. Rose), St. Charles Borromee' and Joliette.

The water plants in the Province of Quebec have a great many taste and odour problems. Many of these do not respond to heavy chlorination and post-ozonation is commonly used to cure these problems. The writer did a survey of ozone installations in the Province of Quebec some years ago. At that time they had some twenty-one installations and seventeen plants had pre and post chlorination with complete treatment and post-ozonation for taste and odour correction.

During the discussion with Beauregard, the above was confirmed. In addition, they do have a number of plants using ozone as the primary oxidant - Sorel, Point Viau, Sherbrook and Joliette but most are post ozonation for taste and odour correction. They like to use free HOCl in the post position for residual when possible. They are using potassium permanganate at Ste. Foy and Granby and powdered activated carbon at Dorval, Pointe Claire, St. Hyacinth and Granby.

Chambly has similar problems to Edmonton. Chlorophenols were identified as a taste and odour problem. Chlorine dioxide was introduced following sedimentation, but before filtration and again to provide a residual during distribution. The new treatment eliminated the chlorophenol taste but caused frequent swampy tastes to occur in the distrubuted water. Tests with ozone showed that it would eliminate this problem. In the interval the upstream industrial source was identified, eliminated and the swampy tastes have now disappeared.

St. Jerome has also encountered swampy tastes following post chlorine dioxide application. The installation of pre-aeration has partially resolved this problem.

Repentigney also installed post chlorine dioxide treatment with continuing taste and odour problems. No resolution was found and the practice has been discontinued.

Joliette was one of the plants which were visited. It is supplied from the L'Assumption River and has a capacity of 10.6 MGPD. The plant had taste and odour problems and utilizes pre-ozonation and conventional flocculation sedimentation using alum plus the flocaide Aqua Floc 464. Then post-ozonation followed by dual media filtration. They add fluoride plus caustic for pH correction and post chlorinate with chlorine dioxide. Water quality is excellent and they have no operating problems.

While at Joliette, I visited a suburb, St. Charles Borromee, which has a small 1.2 MGPD plant treating ground water containing iron. They pre ozonate and filter through dual media - activated carbon on sand and post ozonate with no residual in the distribution system and hence my interest. However, on visiting, I discovered that they have chlorination facilities and provide a 1 PPM residual of chlorine one day every two weeks. The Plant has been in service since June, 1985 with no problems. Prior to Plant construction, the town was supplied by the Joliette Plant so not a clean new system.

The third Plant I visited was located in Laval. The Municipality is located just north of Montreal Island and separated from it by the Riviere des Prairies. On the north side of the Island of Laval runs the Riviere des Mille-Iles. Laval has three treatment plants. The original plant is at Chomedey on the Riviere des Prairies. The original half of the plant uses powdered activated carbon, free HOCl pre and post plus conventional treatment using alum. The new plant is similar but uses no carbon but with pre and post ozonation plus free HOCl for distrubition residuals. They are planning to convert the old plant to pre and post ozonation and abandon the more expensive powdered activated carbon system in the near future. Overall capacity 39 MGPD.

The Point-Viau Plant is also on the Riviere des Prairies and has a capacity of 21 MGPD. It uses pre and post ozonation with chlorine dioxide providing a residual in the distribution system. Turbidity removal is conventional.

The fourth and newest Plant is at St. Rose and has a capacity of 25 MGPD drawn from the Riviere des Mille-Iles. Both the Prairies and the Mille-Iles are part of the mouth of the Ottawa River and run from the Ottawa to the St. Laurence. However, the north shore of the Riviere des Mille-Iles is highly populated, lots of industry and as a result the water quality is much worse than the Riviere des Prairies. T.O.C. on the des Prairies runs 4 mg. per litre in winter to 8 mg. per litre in summer. Mille-Iles' T.O.C. runs 6 mg. per litre in winter and up to 12 mg. per litre in summer. Turbidity averages 4 NTU in winter and up to 80 NTU in spring.

The treatment train consists of pre-chlorine dioxide which is only used in summer, alum feed, Degremont Super Pulsators which have Lamela Separators mounted in the top of a standard pulsator, dual media filtration with a chlorinated backwash for winter use. Filters are Degremont design without troughs and a side flush of water to help carry wastes over the discharge weir. Filters use an air and water wash in sequence. This is followed by ozonation and then storage prior to treatment in activated carbon beds 6.5 ft. deep. Post disinfection by chlorine dioxide. The carbon columns are presently discharging water with a T.O.C. of 2 mg. per litre. They have no regeneration facilities and have not decided how to deal with the issue. The carbon columns are not required for taste and odour removal as chlorine dioxide and ozone provides adequate treatment for that purpose. Plant has been in service since last June and has had few operating problems. The carbon columns are charged with three different carbons - Calgon F400, Hydro Darco 10-30 and a Dutch carbon PKST. They like the F400 best. The PKST is just about exhausted and once it is replaced will postpone the need for regeneration.

Ozone demand is up to 2.5 mg. per litre in part due to the demand of the chlorine dioxide which is converted to chlorate. In order to assure that the post-chlorine dioxide is not also converted, a one hour elapsed time has been designed into the system following ozonation and before the addition of the post chlorine dioxide.

The discussion with Beauregard naturally included chlorine dioxide. Appendix C is a copy of an internal memo with an English translation. The memo applies only to the Montreal District and postpones any further chlorine dioxide installations in the district. The main reason for the action was the fire at Sept-Iles and an explosion at Grenville both due to chlorine dioxide.

They have not experienced any in system generation of hydrocarbon tastes and feel that they only occur at high dose and in the main result from application in the pre-position or inadequate coagulation systems. They have experienced swampy tastes as previously indicated at Chambly and also at both St. Jerome and Repentigney.

They were aware of Sonteimer's comment that chlorine dioxide only generates taste and odours when applied in the pre-position. They had heard from contacts in France of the possible generation of hydrocarbon type odours from reaction of the chlorine dioxide with humic acid and also of the generation of swampy tastes and odours through the reaction of chlorine dioxide plus ammonia plus humic acid.

SUMMARY OF QUEBEC PLANTS

Their practices are similar to those in Alberta. The rates used in flocculation, sedimentation and filtration are comparable. They seem to favour Degremont Pulsators over other solid contact units but that is probably because Degremont is located in Quebec. Unlike Ontario, most water is drawn from rivers, many of which appear to contribute tastes and odours that do notrespond to chlorine. This is not altogether surprising considering the intensive agriculture of the area and the many inadequate waste treatment systems. This has led to the use of all types of taste and odour reducing chemicals and particularly post ozonation for the oxidation of these tastes and odours. The implementation of THM regulations increased the use of pre ozonation and the use of chlorine dioxide to deal with this additional problem.

Chlorine dioxide has however created some taste and odour problems of its own. While no hydrocarbon tastes were reported, they have been getting swampy taste and odours after application of chlorine dioxide.

One U.S. user reported hydrocarbon tastes but exhaustive G.C.M.S. testing has not shown the presence of any hydrocarbons.

CONCLUSIONS

The Operating Licence is used only in Alberta and appears an effective vehicle for the control of drinking water production. Quebec is the only Province to incorporate legally enforceable standards into its drinking water legislation. However, this fact in itself would not appear to impact on the use of the Operating Licence. Alberta and Quebec take very similar approaches to the control of drinking water and are leading Ontario in their control methods.

Technology and practice are very similar in the three jurisdictions. Problems in Ontario tend to differ from in Edmonton while Alberta and Quebec have river sources and Quebec experiences would better relate to problems in Edmonton.

The processes and rates which are currently used do provide for the production of a potable water of acceptable quality but not necessarily free of taste. The operating procedures necessary for taste reduction have still to be determined and it is difficult to predict which of the many approaches might prove most fruitful. Likely candidates are potassium permanganate applied to the intake, ozonation or optimization with relocated points of disinfection.

Increased optimization of the unit processes if based on a reduction of organics may lead to reduced or elimination of tastes and odours particularly if combined with a relocation of the points of application of the feed chemicals. Such optimization, particularly if effluent turbidities can be brought to a 0.1 JTU level has many potential benefits. Higher quality will equate to better removal of bacteria, virus, cysts and therefore improved protection particularly for giardia. Additionally, there would be a reduction in the level of organic and inorganic contaminants and while all of these already meet all current guidelines, the additional protection for the population is gained at little cost.

Optimization to the ultimate does not come easily and is much more difficult and indeed may not be possible when based on a coloured and changing raw water supply which is currently being softened. According to the literature, organics can be absorbed from solution by coagulation preferably at pH about 6.0 for alum or by excess - lime softening at pH about 11.0. In between, removals are poor and seem at their worst around pH 8.0 -9.0.

Staff should investigate the optimum conditions for the removal of organics and the benefits of relocating the points of application for disinfectants. It may be found that softening must be abandoned during taste and odour episodes.

RECOMMENDATIONS

1. The staff of a utility must complete a thorough evaluation of the unit process which is being used. It is only against such a background that the best quality water can be economically produced and that any other process or chemical can be assessed.

2. The potential for contamination is so great that the river bank intake can be used safely only during times of complete ice cover. In the case of a spill or any discharge, rain or snow melt etc. switch to the mid-channel intakes.

3. Once the new mixers are installed in the present program, Plants 1, 2 and 4 will be equipped but not Plant #3. Testing done on our pilot solids contact clarifier indicate that alum added at a mixer ahead of the clarifier produced better results and I would recommend a mixer in the inlet to Plant #3.

4. Regarding the softening at Plants 1 and 2, staff should examine the feasibility of moving the lime addition point into the recarbonation channel with additional mixers to provide additional time for the lime to disolve. Overflow turbidities could be reduced by the use of a coagulant. Jar testing is required to estimate the benefits that can be attained.

5. Present practice calls for the addition of a mixed bag of chemicals to the mixer. The mixer should be used to coagulate the primary coagulant, in this case alum. Never add lime at the same point and I believe the same is true of ammonia. Powdered activated carbon should be added as far upstream from the mixer as it is practicable. The same is true of potassium permanganate, if used. In view of the comments, I would move the chlorine dioxide feed point to after coagulation and the ammonia and chlorine to the filter discharge or even better to the entrance to the storage reservoir or even its discharge depending on in system residuals.

6. Flocculation time in Plants 1 and 2 is critical at maximum flows. The use of a coagulant aid such as activated silica is recommended. If silica is not effective, organic polyelectrolytes could be considered.

7. The rise rate through the tubes is excessive at maximum flow with only alum used as a coagulant. With the addition of a coagulant aid and possibly some weighters such as clay, the rate should be achievable and at the same time reduce overflow turbidity. An objective of 2 NTU is recommended.

8. One of the difficulties in resolving intermittent taste and odour problems is that testing must be done during the event or shortly thereafter. Stored sample water, while better than none, can change. However, one effective treatment for taste and odours is coagulation and optimizing of this segment can be done at any time. I would suggest jar testing at Smith initially without softening and establish the characteristics of the primary coagulants and coagulant aides based on UV extinction, DOC or TOC, THMFP and turbidity objective of O.I NTU. Such a turbidity target has additional benefits. It would significantly increase the protection against giardia cysts. It would reduce the level of potentially harmfull substances in the water supply.

9. Once a taste and odour incident occurs repeat the optimum jar tests without softening and test potassium permanganate, ozone, chlorine dioxide, peroxide, activated carbon and chlorine. Some of this data you probably already have and need not repeat. Permanganate and carbon at least should be tested for contact prior to coagulation. Once a satisfactory treatment is found, then try again with softening and then with disinfection. If you have implemented the results from #8 on a plant scale, you can do the softener tests using plant water following clarification as these jar tests are otherwise difficult to assess.

10. Since you already have sufficient storage at Smith and soon will have at Rossdale to provide residence time for complete disinfection following filtration, this option for taste elimination must be tested. This is particularly important in view of the many comments on chlorine dioxide reactions. Do not ignor the potential of your combined residual in this part of the evaluation program.

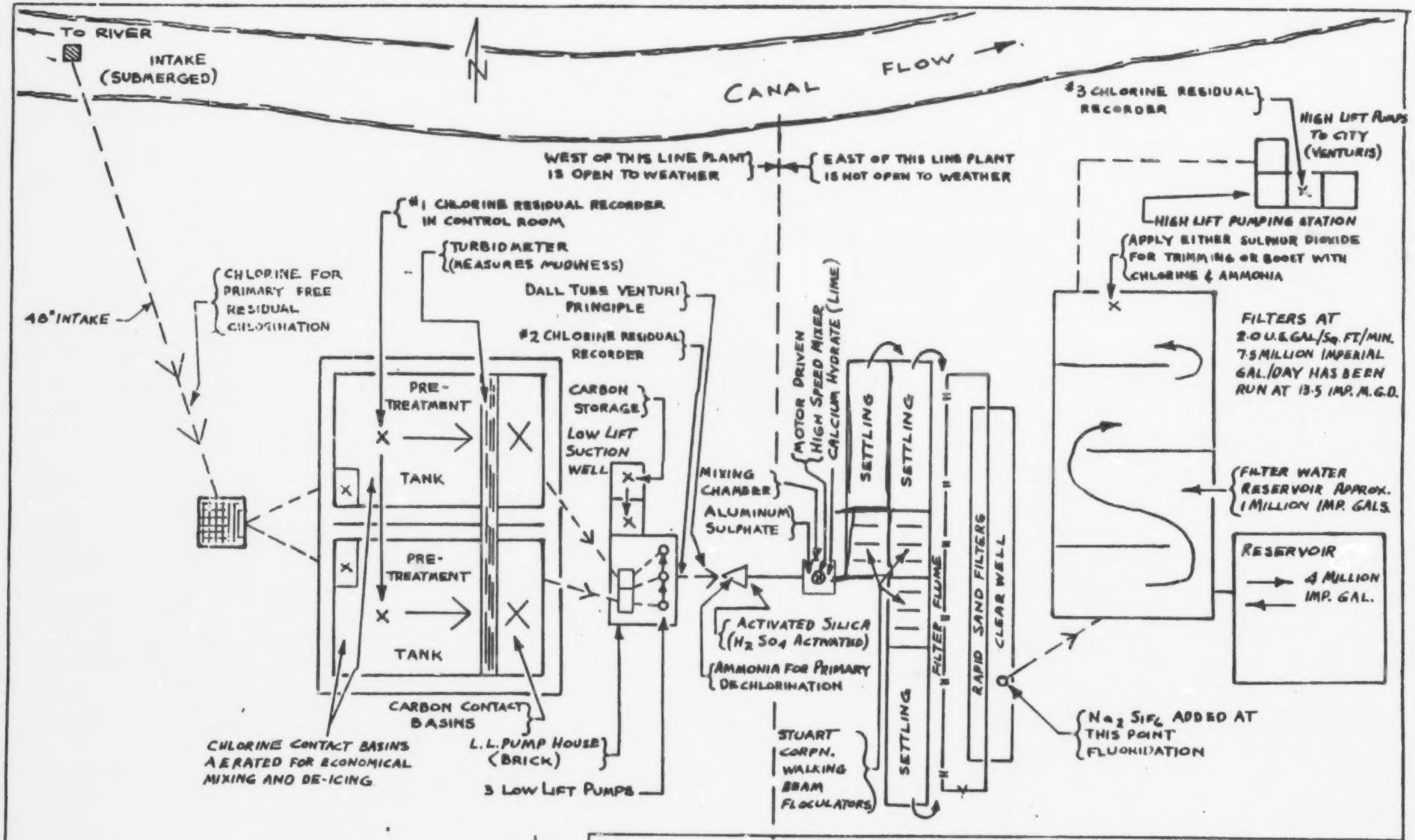
11. If you can find a successful treatment in the tests at Smith and cannot duplicate at Rossdale, then implement a raw water sampling program at Rossdale to determine if intake relocation will resolve the problem - see comments regarding Ottawa. If there is no resolution, the only alternative left is to pipe Smith raw water to Rossdale. This investment can be justified since if the Rossdale Plants are not used, such a main would be needed in any case for the distribution of treated water from the Smith location.

12. Quality Assurance/Quality Control is vital to the operation of any laboratory. The reporting requirements of the Province should insist on a written program. If they do not you should implement your own and participate in some round robin testing of unknowns to test the quality of your results.

13. The benefits of QA/QC extend into the Plant and a similar engineering or Plant QA/QC program is also required. The program should include testing where possible of all flow meters, loss of head gauges, chemical feeders and include tests on parallel units such as at Smith to ensure they do share the flow equally andnot 60-40. This is particularly true of the flows to Plants 1 and 2 which are being severly pushed. Check your rate controllers to ensure filter flows are balanced. Check your residence times and know the degree of short circuiting.

14. It is the responsibility of the regulatory authority to set standards of water quality to protect the public health. It is the responsibility of the utility to produce the best quality water at reasonable price. Much can be done to improve on the standards set and at little cost to the consumer. Also much can be done concerning other unregulated contaminants of concern at the same time.

APPENDIX "A"


TO RIVER
INTAKE (SUBMERGED)
N
CANAL
FLOW
48" INTAKE
CHLORINE FOR PRIMARY FREE RESIDUAL CHLORINATION
WEST OF THIS LINE PLANT IS OPEN TO WEATHER
EAST OF THIS LINE PLANT IS NOT OPEN TO WEATHER
#1 CHLORINE RESIDUAL RECORDER IN CONTROL ROOM
TURBIDMETER (MEASURES MUDINESS)
DALL TUBE VENTURI PRINCIPLE
#2 CHLORINE RESIDUAL RECORDER
PRE-TREATMENT
TANK
PRE-TREATMENT
TANK
CARBON STORAGE
LOW LIFT SUCTION WELL
CARBON CONTACT BASINS
L.L. PUMP HOUSE (BRICK)
3 LOW LIFT PUMPS
CHLORINE CONTACT BASINS AERATED FOR ECONOMICAL MIXING AND DE-ICING
MIXING CHAMBER
ALUMINUM SULPHATE
ACTIVATED SILICA (H_2SO_4 ACTIVATED)
AMMONIA FOR PRIMARY DECHLORINATION
MOTOR DRIVEN HIGH SPEED MIXER
CALCIUM HYDRATE (LIME)
SETTLING
SETTLING
SETTLING
STUART CORPN. WALKING BEAM FLOCULATORS
FILTER FLUME
RAPID SAND FILTERS
CLEAR WELL
Na_2SiF_6 ADDED AT THIS POINT FLUORIDATION
#3 CHLORINE RESIDUAL RECORDER
HIGH LIFT PUMPS TO CITY (VENTURIS)
HIGH LIFT PUMPING STATION
APPLY EITHER SULPHUR DIOXIDE FOR TRIMMING OR BOOST WITH CHLORINE & AMMONIA
FILTERS AT 2.0 U.S. GAL/SQ. FT/MIN. 7.5 MILLION IMPERIAL GAL./DAY HAS BEEN RUN AT 13.5 IMP. M.G.D.
FILTER WATER RESERVOIR APPROX. 1 MILLION IMP. GALS.
RESERVOIR
4 MILLION IMP. GAL.

WATER TREATMENT FACILITIES

PUBLIC UTILITIES COMMISSION - BRANTFORD, ONT.

Nov/10/65					
REV.	DRAWN BY Percival	CHECKED BY H.E.	APPROVED BY RCW	DATE JULY 18/66	SCALE N.T.S.

DRAWING NO W-66-39

APPENDIX "B"

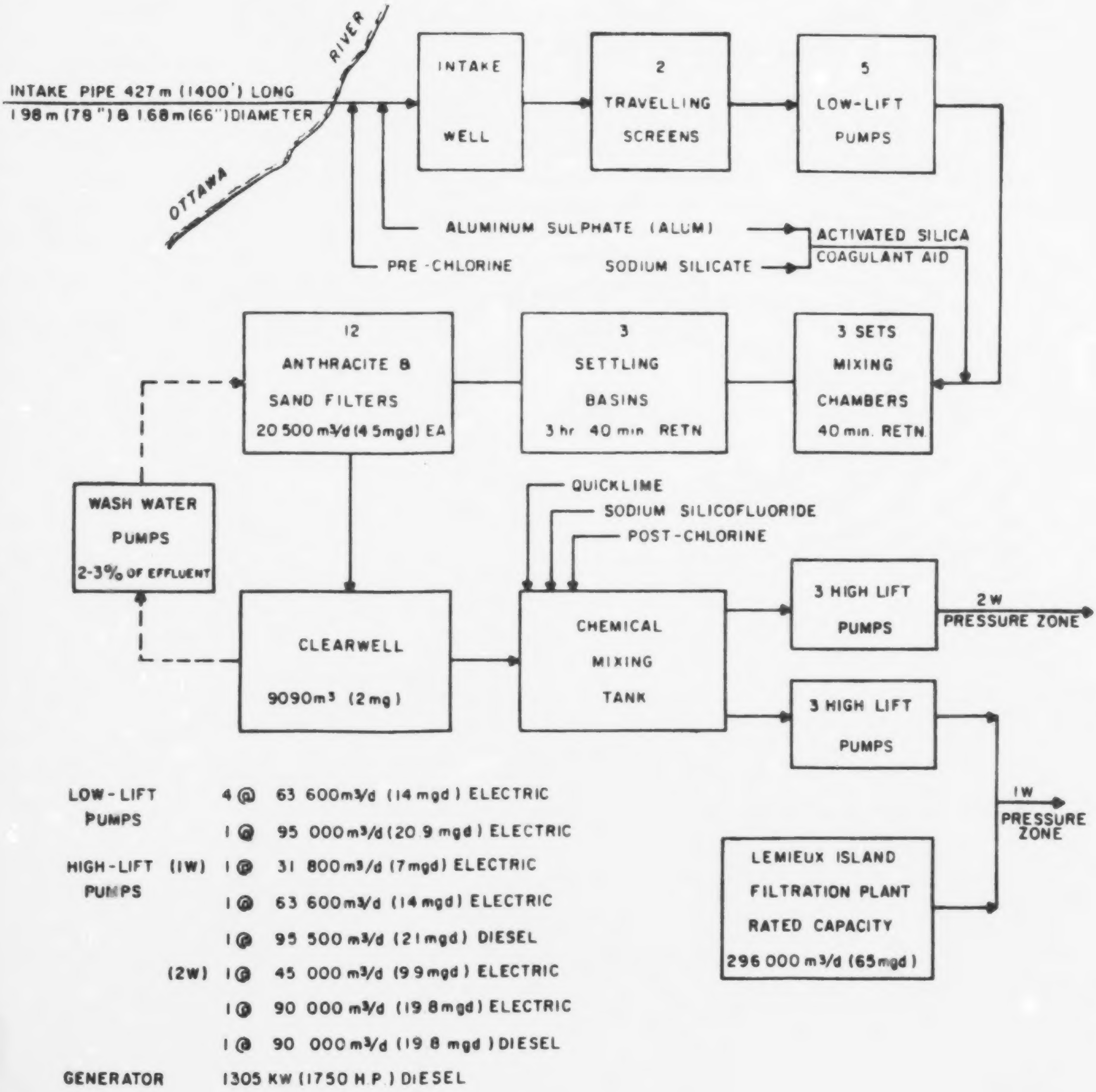
REGIONAL MUNICIPALITY OF OTTAWA-CARLETON

WORKS DEPT. WATER SUPPLY DIVISION

FLOW DIAGRAM

BRITANNIA WATER FILTRATION PLANT AND PUMPING STATION

RATED CAPACITY - 245 000 m³/d (54 mgd)


OTTAWA RIVER
INTAKE PIPE 427 m (1400') LONG
1.98 m (78") & 1.68 m (66") DIAMETER
INTAKE WELL
2 TRAVELLING SCREENS
5 LOW-LIFT PUMPS
ALUMINUM SULPHATE (ALUM)
PRE-CHLORINE
SODIUM SILICATE
ACTIVATED SILICA COAGULANT AID
12 ANTHRACITE & SAND FILTERS 20 500 m³/d (4.5 mgd) EA
3 SETTLING BASINS 3 hr 40 min RETN
3 SETS MIXING CHAMBERS 40 min. RETN.
WASH WATER PUMPS 2-3% OF EFFLUENT
QUICKLIME
SODIUM SILICOFLUORIDE
POST-CHLORINE
CLEARWELL 9090 m³ (2 mg)
CHEMICAL MIXING TANK
3 HIGH LIFT PUMPS
2W PRESSURE ZONE
3 HIGH LIFT PUMPS
1W PRESSURE ZONE
LEMIEUX ISLAND FILTRATION PLANT RATED CAPACITY 296 000 m³/d (65 mgd)

LOW-LIFT PUMPS		4 @	63 600 m³/d (14 mgd) ELECTRIC
		1 @	95 000 m³/d (20.9 mgd) ELECTRIC
HIGH-LIFT PUMPS	(1W)	1 @	31 800 m³/d (7 mgd) ELECTRIC
		1 @	63 600 m³/d (14 mgd) ELECTRIC
		1 @	95 500 m³/d (21 mgd) DIESEL
	(2W)	1 @	45 000 m³/d (9.9 mgd) ELECTRIC
		1 @	90 000 m³/d (19.8 mgd) ELECTRIC
		1 @	90 000 m³/d (19.8 mgd) DIESEL
GENERATOR			1305 KW (1750 H.P.) DIESEL

TRANSLATION

APPENDIX "C"

October 8, 1985

TO: M. Yvan Blouin, Eng.
FROM: Bernard Beauregard, Eng. M.Sc.A.
RE: Chlorine Dioxide

Since the beginning of 1985, the Municipal Section of the Regional Directorate of Montreal, the Ministry of the Environment no longer recommends the installation of disinfection procedures using chlorine dioxide. The water treatment installations effected by this position at this time are in the following Municipalities: Dorval, Terrebonne, Candiac, Repentigney, Sorel and Two Mountains.

The last water purification systems which were authorized to install Chlorine Dioxide were located in Joliette, Ste. Eustache, St.-Jean-Sur Richelieu, Otterbarn Park, Pont-Viau (Laval) and Chambly. In these projects which obtained approval only disinfection by Chlorine Dioxide in post treatment mode have been considered while at the same time disinfection by this oxidant in the pre-treatment mode have been completely eliminated.

The principal reasons which supported this position regarding Chlorine dioxide fall into several catagories and are outlined in the following:

1. The security of the personnel in charge of the maintenance and operation of the equipment in question and for the equipment itself, (fire caused by sodium chlorite at Sept-Isles, explosion in the reactor controls at Grenville).

2. The peace of mind of superintendents and operators in the water treatment plants using chlorine dioxide because of the analysis being carried out on the discharge from the reactor and from the water purification plant daily and at regular intervals, (the complete analysis takes 90 minutes of an operator's time) the equipment for carrying out a complete analysis (amperometric titrator furnished with a platinum electrode and appropriate reagents), the method of analysis itself (a single method to provide a complete analysis for chlorine dioxide, Chlorine, Chlorites and chlorates in the water), of the cost of production of chlorine dioxide (reduction of the addition of acid for the acidification procedure, increasing the addition of Chlorine in those procedures and for Chlorination) and of the Chlorine dioxide residual in the water distribution system (maintaining control of the Chlorites in the water).

3. Tastes and odors could occur due to residuals of Chlorine dioxide of or greater than 0.4 mg./litre.
4. Reasons of health (the reaction of Chlorine dioxide with organic substances in the water results in its reduction to chlorite and the excess chlorine reacting to convert to chlorate).

When a water treatment plant is equipt with pre-ozonation of the raw water, flocculation and sedimentation and adequate filtration, it is superfluous to add chlorine dioxide as a disinfecting agent before sending this water through the distribution system. Besides by article eight of the regulations for potable water (Decree 1158-84, May 16, 1984), the required disinfectant residual in the treated water has been relaxed so that the concentration of free residual chlorine be at least 0.2 mg. per litre 10 minutes after the application, or alternatively a concentration of total residual chlorine of at least 0.2 mg. per litre one hour after application of the chlorine.

When the water distribution system is adequately flushed through the hydrants from the discharge at the water plant to the ends of the system, particularly during the summer season, in order to flush out any deposits and to circulate water into the dead ends of the system in order to prevent their contamination, the persistant biocidal effect of chlorine dioxide would no longer be maintained in the treated water. At the same time, when work is carried out on the distribution system as a result of breaks in the pipeline or for other reasons, it is important to disinfect with chlorine before putting the pipe back in service. This is done by placing chlorine granules in the pipe at the time of replacement and thus prevent the contamination of that part of the system.

The formation of haloforms in potable water by chlorine is reduced when an adequate treatment is applied to the raw water and generally the practice is to pre-ozonate in the treatment chain. Chlorine dioxide is a much stronger oxidant than chlorine for organic substances, the published view that chlorine dioxide does not cause the generation of haloforms does not hold because the excess chlorine which must be introduced for the preparation of chlorine dioxide in order to have a complete reaction (stoichemistry).

A complete study of all chlorine dioxide systems installed with or without authorization by the Ministry of the Environment in the Montreal Region with complete analysis, photographs, descriptions and system operations must be carried out this winter when other demands are less pressing.

Beauregard

Draft for Comment

WATER QUALITY ASSESSMENT

a task report for

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

by

Steve E. Hrudey & Associates Ltd.

July 1986

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Appendix A Historical Water Quality in the North Saskatchewan River at Edmonton and Upstream

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1. INTRODUCTION

This report addresses tasks 2, 5a and 5b of the overall study Terms of Reference (provided in Appendix A of the Overview Report in Volume 1). Specifically, water quality of the source, the North Saskatchewan River, and the treated water distributed to Edmonton consumers is critically assessed.

Comparisons are made with existing water quality criteria and with comparable situations in other jursidictions. Issues of particular concern to the Edmonton water supply are reviewed in more detail.

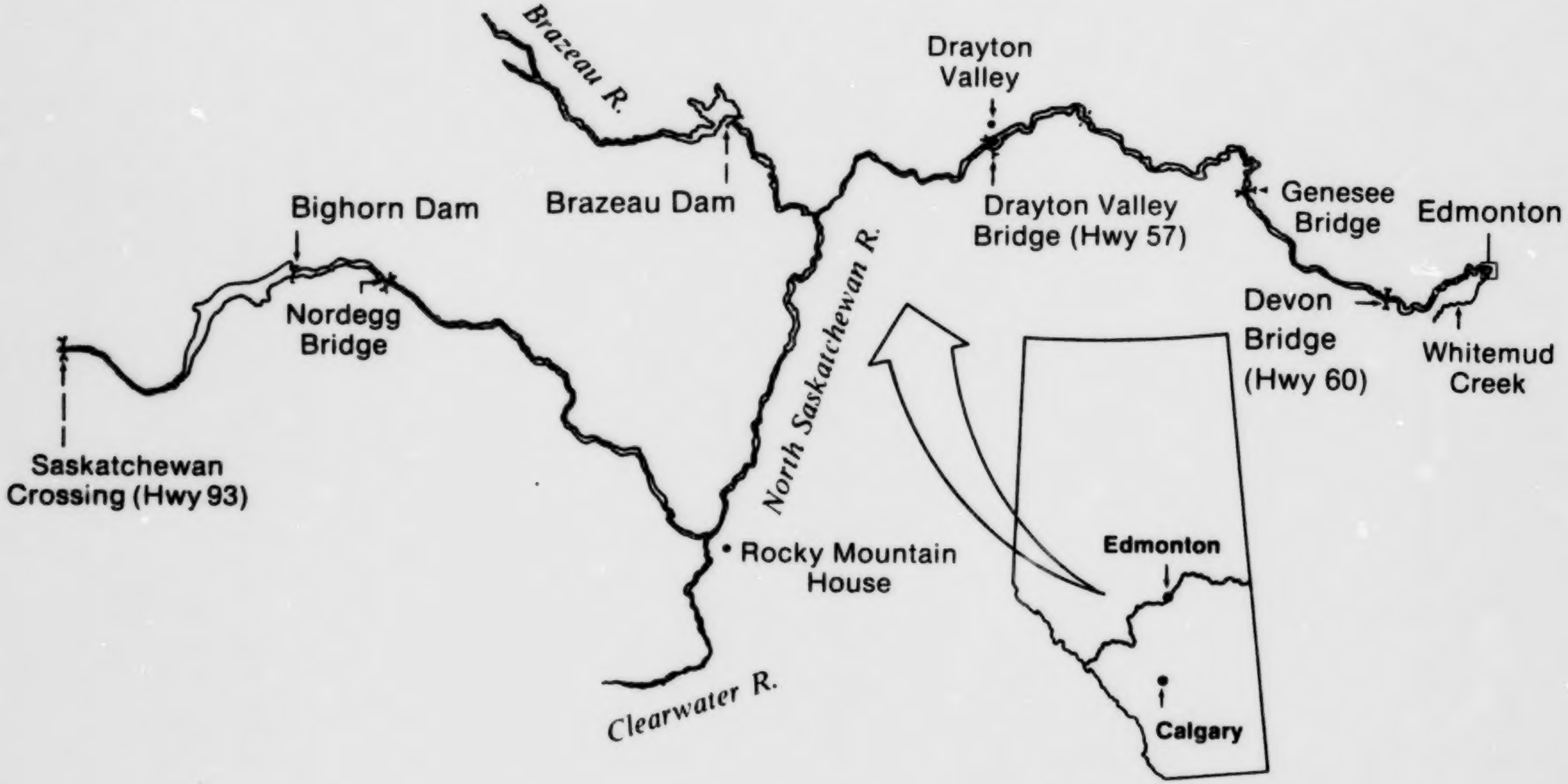
2. Raw Water Supply - North Saskatchewan River

2.1 Description

The North Saskatchewan River rises from the Saskatchewan Glacier in the Columbia Icefields approximately 500 km to the west of Edmonton (Figure 1.1). Glacier melt typically provides approximately 5% of the river's flow in winter and up to about 50% of the flow in July[1]. Major tributaries include the Clearwater, Brazeau and Nordegg Rivers. In winter, on average (1972 - 1982), these contribute approximately 6%, 36% and <1% of the mainstream flows, respectively. In July, on average, they contribute approximately 13%, 30% and 3% of the mainstream flows, respectively.

The glacial melt is a high quality source, but the water provided by the tributaries, which drain forested and muskeg

Figure 1.1. The North Saskatchewan River Basin Upstream of Edmonton.



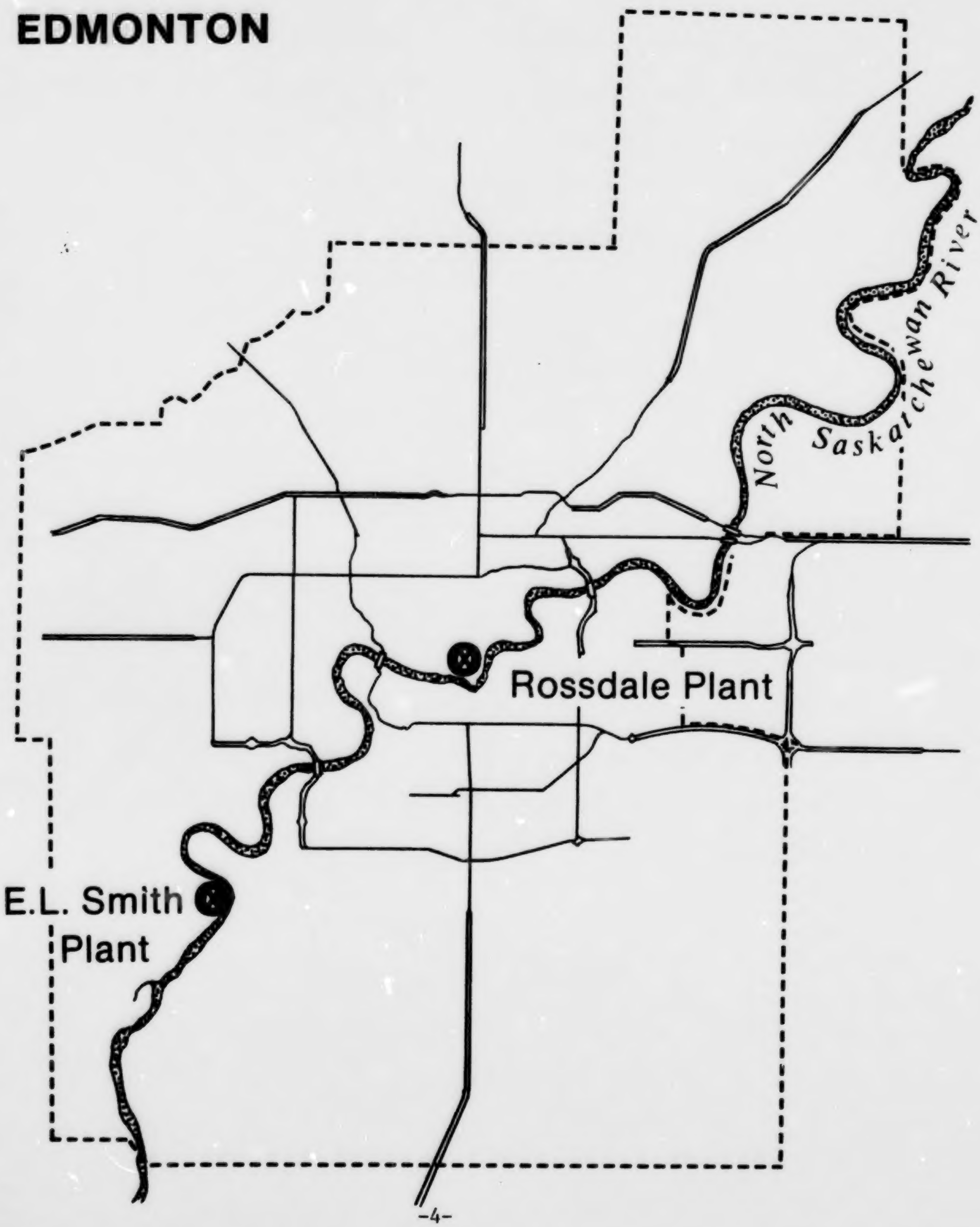
areas, carries a higher organic loading. Several smaller streams, often with even higher organic content, also join the North Saskatchewan River upstream of Edmonton.

Since the introduction of flow regulation by the Brazeau (1963) and Big Horn (1972) dams, minimum flows in the river have been maintained. Monthly average flows are generally above 90 m^3/s a level which does not pose any quantity contraint on water withdrawals by Edmonton.

In travelling from the Rockies to Edmonton, the river traverses three geological zones[2]. In the mountain region, the geology consists of older Pre-Cambrian and Paleozoic formations consisting primarily of sandstone, shale, limestone and dolomite. The foothills region, formed in the Mesozoic era, consists of sandstone, shale and coal formations. Finally, the Edmonton region, also of the Mesozoic era, consists of sandstone, shale, coal and minor bentonite formations.

Edmonton's drinking water is produced from two major facilities, Rossdale and E.L. Smith. The first is located northeast of the 105 Street bridge and the second across the river and southwest from Terwillegar Park (Figure 1.2).

Figure 1.2. Location of Edmonton Water Treatment Plants.



2.2 Water Quality Criteria

2.2.1 Alberta Relative to Other Jurisdictions

The Alberta Clean Water Act provides the Minister of Environment with a wide range of powers to prevent pollution of surface waters. In practice, any facility which is capable of causing water pollution is required to obtain a "permit to construct" before commencing construction. Such facilities are then required to obtain a "license to operate" prior to commencing operation. The latter can prescribe quality and quantity constraints on any discharges to surface waters. These levels are decided, in part, by reference to the Alberta Surface Water Quality Objectives (ASWQO)[3]. These objectives do not, in themselves, have the force of law. However, discharge levels, set with the ASWQO in mind, will have regulatory force once incorporated into the license to operate.

The ASWQO have been derived from experience elsewhere and many of the values are common to other jurisdictions.[4] A comparison of ASWQO with other Canadian and U.S. criteria is summarized in Table 1. The number of parameters covered by this table is small relative to the number of pollutants which are currently receiving attention. Considering that there are over 7 million chemical substances registered with the American Chemical Society CAS Registry, the task of accounting for all possible concerns to water quality is staggering.

Criteria have only been developed for a limited number of substances. Experience has shown that these warrant attention. Very few specific organic chemicals, including pesticides, have had specific surface water quality criteria adopted. More attention has been devoted to specifying allowable levels for such chemicals in treated water supplies and these will be discussed in section 3.2.

The status of the ASWQO will be reviewed by considering each parameter from Table 1, in turn. Only those parameters from the ASWQO which are relevant to criteria for a raw water supply are considered, in keeping with the terms of reference of this study. Table 1 includes all parameters either specified by other Canadian jurisdictions for raw water sources or parameters specified by the ASWQO but not other jurisdictions.

Table 1. Comparison of Alberta Surface Water Quality Objectives with Those of Other Jurisdictions (raw water supply)

Parameter	Alberta (1977)[3]	Manitoba (1983)[5] (provisional)	Quebec (1984)[6]	Environment Canada[7] (1979-1983)	U.S.E.P.A.[8,9] (I:1976, II:1980)
Antimony	not specified	0.0002 mg/L	0.1 mg/L (b)	not specified	0.146 mg/L
Arsenic	0.01 mg/L	0.05 mg/L	0.05 mg/L (b)	0.05 mg/L	(I) 0.000002 mg/L (II) 0.05 mg/L
Bacteriology					
Fecal Coliforms	90 percentile: 1000 per 100mL	90 percentile: 10 per 100 mL (a)	absent (b) 10 -100 (a) >100 (d)	not specified	not specified (for raw water sources)
Total Coliforms	90 percentile: 5000 per 100mL	90 percentile: 100 per 100 mL (a)	10 (b) 100-1000 (a) >1000 (d)	not specified	not specified (for raw water sources)
Barium	1.0 mg/L	1.0 mg/L	1.0 mg/L	not specified	(I) 1.0 mg/L
Boron	not specified	5.0 mg/L	5.0 mg/L	not specified	not specified (for raw water sources)
Cadmium	0.01 mg/L	0.005 mg/L	0.005 mg/L (b) 0.01 mg/L (b) 0.02 mg/L (e)	0.01 mg/L (d) 0.02 mg/L (e)	0.01 mg/L
Carbon Chloroform Extract	0.2 mg/L	not specified	not specified	not specified	not specified
Chromium	0.05 mg/L	0.05 mg/L	0.05 mg/L	0.1 mg/L	(II) 0.05 mg/L (I) 0.05 mg/L (Cr+6)
Copper	0.02 mg/L	1.0 mg/L	1.0 mg/L	0.25 mg/L	1.0 mg/L
Colour	<30 + natural	not specified	not specified	not specified	(I) 75 units (Pt-Co)
Cyanides	0.01 mg/L	0.2 mg/L	0.2 mg/L	not specified	(II) 0.2 mg/L
Halides					
Chlorides	site specific	250 mg/L	250 mg/L	not specified	not specified
Fluorides	1.5 mg/L	1.5 mg/L	1.5 mg/L	not specified	not specified
Iron	0.3 mg/L	0.3 mg/L	0.3 mg/L	not specified	(I) 0.3 mg/L
Lead	0.05 mg/L	0.05 mg/L	0.05 mg/L (b,a)	0.25 mg/L (d) 0.05 mg/L (b)	0.05 mg/L
Manganese	0.05 mg/L	0.05 mg/L	0.05 mg/L	0.05 mg/L	(I) 0.05 mg/L

(continued)

Table 1 continued

Parameter	Alberta (1977)[3]	Manitoba(1983)[5] (provisional)	Quebec(1984)[6]	Environment Canada[7] (1979-1983)	U.S.E.P.A.[8,9] (I:1976,II:1980)
Mercury	0.0001 mg/L	0.001 mg/L	0.001 mg/L	0.001 mg/L	(I)0.002 mg/L (II)0.00014 mg/L
Methyl Mercaptan	0.05 mg/L	not specified	not specified	not specified	not specified
Methylene Blue Active Substances	0.5 mg/L	not specified	not specified	not specified	not specified
Nickel	not specified	not specified	0.25 mg/L(b,d)	0.25 mg/L(d)	(II)0.0134 mg/L
Nitrogen					
Nitrate+nitrite	1 mg/L	10 mg/L	10 mg/L	not specified	(I)10 mg/L
Odor	TON < 8 (20°C)	not specified	not specified	not specified	not specified
Oil & Grease	absent no sheen	not specified	not specified	not specified	(I)absent
pH	6.5 - 8.5	6.5 - 8.5	6.5 - 8.5	not specified	(I)5 - 9
phenolics	0.005 mg/L	0.002 mg/L	not specified	not specified	(I)0.001 mg/L (II)0.3 mg/L (phenol)
Radionuclides					
Cesium-137	not specified	50 Bq/L	not specified	not specified	not specified
Iodine-131	not specified	10 Bq/L	not specified	not specified	not specified
Radium-226	0.11 Bq/L	1 Bq/L	not specified	not specified	not specified
Strontium-90	0.37 Bq/L	10 Bq/L	not specified	not specified	not specified
Tritium	not specified	40,000 Bq/L	not specified	not specified	not specified
Selenium	0.01 mg/L	0.01 mg/L	0.01 mg/L(b,a) 0.25 mg/L(e)	0.05 mg/L(c) 0.25 mg/L(e)	0.01 mg/L
Silver	0.05 mg/L	0.05 mg/L	0.05 mg/L(b,a) 0.2 mg/L(d)	0.05 mg/L(c) 0.2 mg/L(e)	0.05 mg/L
Sulphates	site specific	500 mg/L	500 mg/L	not specified	not specified
Turbidity	<25 JTU + natural	5 NTU	5 NTU(b)	not specified	not specified (for raw water sources)

Table 1 continued

Parameter	Alberta (1977)	Manitoba(1983) (provisional)	Quebec(1984)	Environment Canada (1979-1983)	U.S.E.P.A. (I:1976,II:1980)
Uranium	not specified	0.02 mg/L	0.02 mg/L	0.02 mg/L	not specified
Zinc	0.05 mg/L	5.0 mg/L	5.0 mg/L(b) 10.0 mg/L(a,d)	5.0 mg/L(c) 10.0 mg/L(e)	5 mg/L

(a)disinfection only (b)use without treatment (c)sand filtration only (d)complete treatment: flocculation-coagulation, sedimentation, filtrat and disinfection (e)conventional complete treatment plus lime precipitation and activated carbon

2.2.1.1 Antimony. This metal is not included in the ASWQO. The other values quoted span a wide range from 0.0002 mg/L (Manitoba, 1983, provisional) to 0.1 mg/L (Quebec, 1984). The low value corresponds to the objective level from the 1978 Canadian Drinking Water Guidelines[10]. However, that source[10] did not elect to specifiy a maximum acceptable concentration (MAC) for antimony because there were insufficient data to establish a health based MAC. Available information at that time (1978) suggested that the intake of antimony from drinking water was insignificant relative to intake from food. The U.S.Environmental Protection Agency[11] also deferred action on setting a recommended maximum contaminant level (RMCL) for antimony in their 1985 drinking water regulatory proposals on the grounds of limited evidence for potential risk associated with drinking water exposure to antimony.

Alberta Environment should monitor evidence as it is produced by the Department of National Health and Welfare and the U.S.E.P.A. to determine the need for including antimony in an updated version of the ASWQO. At present, there is no evidence from other agencies to suggest an urgent need to include antimony in the ASWQO. For current purposes, the North Saskatchewan River will be judged relative to Manitoba objectives which relate to waters which could be withdrawn for water supply with disinfection only.

2.2.1.2 Arsenic. The ASWQO specify a level of 0.01 mg/L which is 1/5 of the levels proposed by other jurisdictions with the exception of the 1980 E.P.A. value. That exception corresponds to a carcinogenic risk estimate for one in a million lifetime risk of cancer arising from arsenic exposure. The value obtained has not been adopted for the regulatory level under the U.S. Safe Drinking Water Act. The regulatory proposal[11] for arsenic was a recommended maximum contaminant level (RMCL) of 0.05 mg/L. This was based upon the level recommended by the U.S. National Academy of Sciences and data which suggest that arsenic may be an essential nutrient. Consequently, the ASWQO are more stringent than the effective levels adopted by the other jurisdictions surveyed.

2.2.1.3 Bacteriology. Criteria have been specified for fecal coliforms and total coliforms. The levels adopted by Alberta for sources regarded as acceptable for raw water sources are higher than those proposed by other jurisdictions. They are consistent with levels proposed by the Canadian Drinking Water Guidelines[10] for water sources which could be accepted provided they received complete treatment (coagulation-flocculation, sedimentation, filtration and disinfection) supplemented by auxiliary treatment (pre-chlorination or pre-sedimentation or equivalent and post-chlorination). Given the generally good water quality which is available in Alberta and emerging constraints upon disinfection practice which will be discussed in section 3.6, the ASWQO for fecal and total coliform levels in potential raw water sources are too high. They should be reviewed in light of Alberta's needs and general water quality conditions to encourage selection of better raw water quality for water supplies. The low levels prescribed by the Manitoba and Quebec objectives apply to waters which could be used for drinking water after only disinfection.

2.2.1.4 Barium. The level of 1.0 mg/L specified by the ASWQO is completely consistent with levels proposed by others and does not seem to be a cause for any concern at this time.

2.2.1.5 Boron. Currently no level is specified in the ASWQO for boron. Other jurisdictions have adopted a level of 5 mg/L which will be used to assess the North Saskatchewan River quality. Alberta Environment should review the occurrence of boron in Alberta surface waters and determine if there is any need for including a boron value in the ASWQO.

2.2.1.6 Cadmium. The ASWQO level of 0.01 mg/L is consistent with proposals from other jurisdictions for waters which will receive some treatment prior to distribution. It is double the level specified for waters to be used without treatment. Although the latter practice is not generally desirable, Alberta Environment should review their cadmium objective to determine if recent literature provides any basis for lowering the current value.

2.2.1.7 Carbon Chloroform Extract. No other jurisdiction provides a quality objective for this parameter. It is now outdated and has been dropped from the 1985 edition of Standard Methods[12]. Accordingly, it should be dropped from the ASWQO.

2.2.1.8 Chromium. The ASWQO of 0.05 mg/L is consistent with other jurisdictions and does not appear to warrant any concern.

2.2.1.9 Copper. The ASWQO of 0.02 mg/L is well below (1/12 to 1/50) levels specified by other jurisdictions. This likely reflects the multiple use nature of the ASWQO and the criterion is controlled by the need to protect fish. Consequently, in terms of drinking water quality, the ASWQO are more than adequate, based upon commonly accepted values.

2.2.1.10 Colour. The ASWQO calls for less than 30 units (method not specified) of colour increase above natural background. Other jurisdictions have not addressed this parameter, except for EPA (1976)[8] which called for less than 75 units (Pt-Co) total colour in waters to be extracted for water supply. Both values would pose some treatment challenges to smaller municipalities. Although natural values can exceed such levels, highly colored waters should be avoided for water sources when other choices are available. The ASWQO should reflect that reality. As well, the ASWQO should discourage any waste discharge which could give rise to colour increases higher than the current Canadian Drinking Water Guideline[10] of 15 true colour units.

2.2.1.11 Cyanides. The ASWQO of 0.01 mg/L is 1/50 of levels proposed by other jurisdictions for drinking water sources. This level is likely set for the protection of aquatic life and appears to be more than adequate for drinking water protection based upon currently accepted values.

2.2.1.12 Chlorides. No number is actually set for chorides. Rather, a site specific assessment is suggested based upon local conditions. This is necessary because of the wide range of natural chloride levels which can be encountered. The value of 250 mg/L used by other jurisdictions will be used to judge the North Saskatchewan River water quality.

2.2.1.13 Fluorides. The ASWQO specify a level of 1.5 mg/L for fluorides which is consistent with levels proposed by other jurisdictions who have adopted a fluoride objective.

2.2.1.14 Iron. The ASWQO specify a level of 0.3 mg/L for iron which is consistent with levels proposed by other jurisdictions who have adopted an iron objective.

2.2.1.15 Lead. The ASWQO specify a level of 0.05 mg/L for lead which is consistent with levels proposed by other jurisdictions who have adopted a lead objective.

2.2.1.16 Manganese. The ASWQO specify a level of 0.05 mg/L for manganese which is consistent with levels proposed by other jurisdictions who have adopted a manganese objective.

2.2.1.17 Mercury. The ASWQO specify a level of 0.0001 mg/L for mercury which is 1/10th to 1/20th of the levels adopted by most other jurisdictions. This lower value likely reflects the multiple objective nature of the ASWQO and the need to protect fish from mercury. Because mercury can be bioconcentrated, there is a concern with mercury contamination of fish intended for human consumption (section 2.6.2).

2.2.1.18 Methyl Mercaptan. The ASWQO specify a level of 0.05 mg/L for methyl mercaptan while no other jursidiction proposes an objective for this chemical. The choice of this chemical for a specific objective requires justification because it is only one of a family of odorous sulphur compounds which may occur in water. Its odor threshold in water is reported to range from 0.0011 mg/L to 0.041 mg/L[13] but the closely related compound, ethyl mercaptan, causes taste and odor at 0.00019 mg/L[13]. Consequently, similar

2.2.1.15 Lead. The ASWQO specify a level of 0.05 mg/L for lead which is consistent with levels proposed by other jurisdictions who have adopted a lead objective.

2.2.1.16 Manganese. The ASWQO specify a level of 0.05 mg/L for manganese which is consistent with levels proposed by other jurisdictions who have adopted a manganese objective.

2.2.1.17 Mercury. The ASWQO specify a level of 0.0001 mg/L for mercury which is 1/10th to 1/20th of the levels adopted by most other jurisdictions. This lower value likely reflects the multiple objective nature of the ASWQO and the need to protect fish from mercury. Because mercury can be bioconcentrated, there is a concern with mercury contamination of fish intended for human consumption (section 2.6.2).

2.2.1.18 Methyl Mercaptan. The ASWQO specify a level of 0.05 mg/L for methyl mercaptan while no other jursidiction proposes an objective for this chemical. The choice of this chemical for a specific objective requires justification because it is only one of a family of odorous sulphur compounds which may occur in water. Its odor threshold in water is reported to range from 0.0011 mg/L to 0.041 mg/L[13] but the closely related compound, ethyl mercaptan, causes taste and odor at 0.00019 mg/L[13]. Consequently, similar compounds could be more critical depending upon the rationale for inclusion in the ASWQO.

In any case, no monitoring data for this compound have been located so it is not possible to judge North Saskatchewan River data against this objective. However, this compound is not considered a likely problem for a well oxygenated river like the North Saskatchewan unless a specific spill event caused a major input to the river.

2.2.1.19 Methylene Blue Active Substances. The ASWQO specify a value of 0.5 mg/L for methylene blue active substances. No other jurisdiction proposes a value for this parameter. It is an empirical measure for anionic surfactants which was developed to provide a routine analysis for detergents in water. This was particularly relevant when non-biodegradable detergents were still widely in use. Although the method is relatively simple and can be quite precise, it is not specific for any particular compound[12]. Likewise, it does not respond to non-ionic or cationic surfactants so that it cannot give a total measure of surfactants. The method has not been used in recent monitoring of the North Saskatchewan River.

2.2.1.20 Nickel. The ASWQO do not specify a level for nickel. Levels from 0.0134 to 0.25 mg/L have been proposed by other jurisdictions. The lower level proposed by E.P.A.(1980) has not been reflected in their Safe Drinking Water regulation proposals[11] where the E.P.A. opted to not regulate nickel. They did however propose a guidance level of 0.15 mg/L for nickel. Alberta Environment should review the current literature and available data on Alberta waters to determine if there is a need for a ASWQO value for nickel. North Saskatchewan River data will be compared with the other proposed levels.

2.2.1.21 Nitrogen: Nitrate+Nitrite. The ASWQO propose a level of 1 mg/L for total nitrogen in all forms including organic nitrogen and ammonia. Other jurisdictions propose an objective of 10 mg/L for just nitrate+nitrite nitrogen. Consequently, the ASWQO level is much more stringent than levels used by other jurisdictions.

2.2.1.22 Odour. The ASWQO propose a Threshold Odour Number limit of 8 (at 20°C) while no other jurisdiction proposes an odor objective on raw water for drinking water supply.

2.2.1.23 Oil and Grease. The ASWQO propose that oil and grease should be substantially absent (no sheen) from surface waters. This is consistent with the E.P.A. (1976)[8] criterion. Unfortunately, most of the data which has been collected has used the partition-gravimetric method[12] of analysis which gives virtually meaningless results for river samples.

The ASWQO refers to "ether solubles" which is an obsolete parameter. Alberta Environment should specify a gas chromatographic or infrared absorbance method for data collection relative to this objective. The available data on the North Saskatchewan River, other than recent monitoring programs which will be discussed in sections 2.5 and 2.6, is unreliable because of the analytical method used.

2.2.1.24 pH. The ASWQO specify an objective pH range from 6.5 to 8.5 which is consistent with requirements for other jurisdictions.

2.2.1.25 Phenolics. The ASWQO specify an objective of 0.005 mg/L for phenolics. Manitoba (1983, provisional)[5] proposes 0.002 mg/L for waters which which could be consumed after only chlorine disinfection. The U.S.E.P.A (1976)[8] proposes 0.001 mg/L for the compound phenol, which is one of the many possible phenolic compounds. The difference in compounds specified likely accounts for some of the difference in objective numbers. Many complex natural molecules include phenolic components in their molecular structure. Some of these presumably can respond to the routine colorimetric analysis used.[12]

Alberta Environment should review the analytical methods available to support the phenolics objective and determine if a more specific analytical method would provide a better basis for this objective.

2.2.1.26 Radionuclides. The ASWQO only specify levels for the two radionuclides radium-226 (at .11 Bq/L) and strontium-90 (at 0.37 Bq/L). These levels are 1/10th or less of the Manitoba[5] objectives which are derived from the Canadian Drinking Water Guidelines[10]. Alberta Environment should consider adding the radionuclides cesium-137, iodine-131 and tritium in order to be consistent with the Canadian Drinking Water Guidelines[10] or their anticipated update.

2.2.1.27 Selenium. The ASWQO propose a level of 0.01 mg/L for selenium which is 1/5 of the Environment Canada[7] proposal but is otherwise consistent with other jurisdictions.

2.2.1.28 Silver. The ASWQO of 0.05 mg/L for silver is consistent with other jurisdictions and does not appear to warrant any concern.

2.2.1.29 Sulphates. The ASWQO do not specify a value for sulphates because of site specific variations in the natural levels of this parameter. In any case, normal levels are well below the objective level of 500 mg/L proposed by other jurisdictions.

2.2.1.30 Turbidity. The ASWQO specify that turbidity levels should not be increased more than 25 JTU above background. Other jurisdictions are calling for turbidity of less than 5 NTU for waters to be used for water supply without treatment or with disinfection only. The turbidity level proposed by the ASWQO would not pose any problem to a complete water treatment process. However, the allowable increase of 25 JTU might complicate water treatment for a small water supply and this objective should be reviewed in that light. As well, the objective should likely be expressed in NTU because nephelometry provides a more reliable basis of measurement than the visual procedure used for JTUs.

2.2.1.31 Uranium. The ASWQO do not specify a level for uranium. The North Saskatchewan River data will be reviewed relative to the 0.02 mg/L level specified by other jurisdictions. Alberta Environment should review monitoring data on Alberta rivers to determine if there is a case for including a uranium objective in the ASWQO.

2.2.1.32 Zinc. The ASWQO specify a zinc level of 0.05 mg/L which is 1/100th of the levels proposed by other jurisdictions. The documentation for the low ASWQO level suggests that it is set because zinc "produces an undesirable taste, and can be synergistic". The low levels adopted in the ASWQO cannot be based upon a taste criterion according to the rationale adopted by other jurisdictions. Alberta Environment should review the criterion for zinc to determine if danger to aquatic life or some other valid concern can justify the low value currently in place.

2.1.1.33 Summary. Overall, the ASWQO are quite comparable to water quality criteria used by other jurisdictions. In a few cases, ASWQO levels were either not provided or were higher than values used by others. In these cases, a review of the ASWQO has been recommended and in specific cases lower values are likely required. In other cases, the ASWQO were lower than values used by other jurisdictions often reflecting the multiple use nature of the ASWQO. An updating of the ASWQO is necessary to make it consistent with current analytical procedures.

2.2.2 The North Saskatchewan River as Judged Against the Alberta Surface Water Quality Objectives.

In Section 2.1.1, the ASWQO were critcally compared with water quality criteria used by other jurisdictions and the ASWQO were found to provide water quality targets which compare favorably with others. In this section, recent monitoring data for the North Saskatchewan River will be compared against those criteria. In cases where the ASWQO do not provide a value, or provide a value higher than relevant values from elsewhere, the stricter value is used in the comparison.

The North Saskatchewan River data is provided from Alberta Environment monitoring at Devon[14] between January 1982 and December, 1984. Where specific parameters are not available from this data base, they are taken from 1984 monitoring by the City of Edmonton at the E.L. Smith plant.

Water quality in the North Saskatchewan River does not change significantly between Devon and the E.L. Smith plant. Because water quality does change substantially between the E.L. Smith plant and the Rossdale plant under the influence of urban runoff, that issue will be dealt with separately in section 2.6.

The comparisons are plotted in Figures 2.1 to 2.29. These indicate that for the majority of parameters typical North Saskatchewan River water quality coming into Edmonton is good relative to the Alberta Surface Water Quality Objectives or alternate objectives from other jurisdictions where the latter are lower. Certain specific parameters warrant comment.

A single high value for cadmium exceeded the Manitoba criterion which is equivalent to the Canadian Drinking Water Guideline level for treated drinking water and is equal to 50% of the Alberta Surface Water Quality Objective. The only value exceeding the objective was one out of 30 samples taken between 1982 and 1986. A total of 20 out of the 30 samples during this period were below the analytical detection limit for this metal. Also, the monitoring is done for total metal (including suspended materials) rather than dissolved. Suspended metal will normally be well removed with turbidity in the water treatment process. Considering these factors, the quality of the North Saskatchewan River with respect to cadmium is not a problem.

The maximum value for chromium was 0.044 mg/L for a single sample in 1982. Values since 1983 have been consistently below 0.006 mg/L and they are frequently below the detection limit of 0.001 mg/L.

The maximum value for copper was 0.016 mg/L for a single sample in 1982. Values since 1983 have been consistently below 0.004 mg/L and they are frequently below the detection limit of 0.001 mg/L. The ASWQO for copper is substantially lower than the Canadian Drinking Water Guideline because the former is set for the protection of fish which are more sensitive to copper than humans.

The maximum value for iron was 1.04 mg/L in 1982. Occasional high values have been observed since then. The iron content of the North Saskatchewan River apparently does occasionally exceed the ASWQO. Because iron is a consideration in drinking water strictly for aesthetic reasons and it does not pose any problem with the treatment processes employed in Edmonton, the iron values are not a major concern as a measure of source water quality for Edmonton.

The maximum value for manganese was 0.087 mg/L in 1982. Occasional high values have been observed since then. The manganese content of the North Saskatchewan River does occasionally approach up to 70% of the ASWQO although that one maximum value was the only value above the ASWQO for 1982 to 1985. Because manganese is a consideration in drinking water strictly for aesthetic reasons and it does not pose any problem with the treatment processes employed in Edmonton, the manganese values are not a major concern as a measure of source water quality for Edmonton.

The values for mercury appear high as depicted but they are, in fact, below the detection limit which, in this case, coincides with the ASWQO. The latter is effectively set as low as can be reliably measured on a routine basis.

A single high value for nickel exceeded the U.S. Environmental Protection Agency surface water quality criterion for nickel. That agency has not specified a drinking water guideline for nickel, nor have the Canadian Drinking Water Guidelines. Apart from the single high value only 3 other samples out of 30 taken between 1982 and 1986 are more than half the objective level and 5 were below the analytical detection limit. These values were also reported for total nickel. Considering these factors, the quality of the North Saskatchewan River with respect to nickel is not a problem.

The values observed for phenolics at Devon are considerably higher than observed by the E.L. Smith plant where results on raw water were consistently below a detection limit of 0.002 mg/L in 1984. The reasons for this discrepancy are not clear, but may relate to some localized source in the area which is affecting the Devon site but is insufficient to affect downstream water quality.

The Manitoba objective for turbidity is based on waters which could be distributed after only disinfection. Although for much of the year the North Saskatchewan River could meet the objective, use of surface water without filtration is becoming recognized as an undesirable practice (see Logsdon report, this volume). Treatment without filtration would never be contemplated for Edmonton so the turbidity values do not pose any significant problems for Edmonton.

The uranium values are taken from the 1984 raw water monitoring at the E.L. Smith plant. The maximum value is apparently still below a detectable limit but that result was reported with a higher limit. This value, even if taken to be at a detectable level, is below the maximum acceptable concentration for treated water.[10]

The zinc value for the ASWQO was noted in Table 1 to be much lower than values used by other jurisdictions for drinking water sources. Thus, although the maximum values measured at Devon approach the ASWQO, there does not appear to be much significance attributable to that. For comparison, the Canadian Drinking Water Guideline[10] for treated water calls for less than 5 mg/L.

Fig. 2.1 to 2.4. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

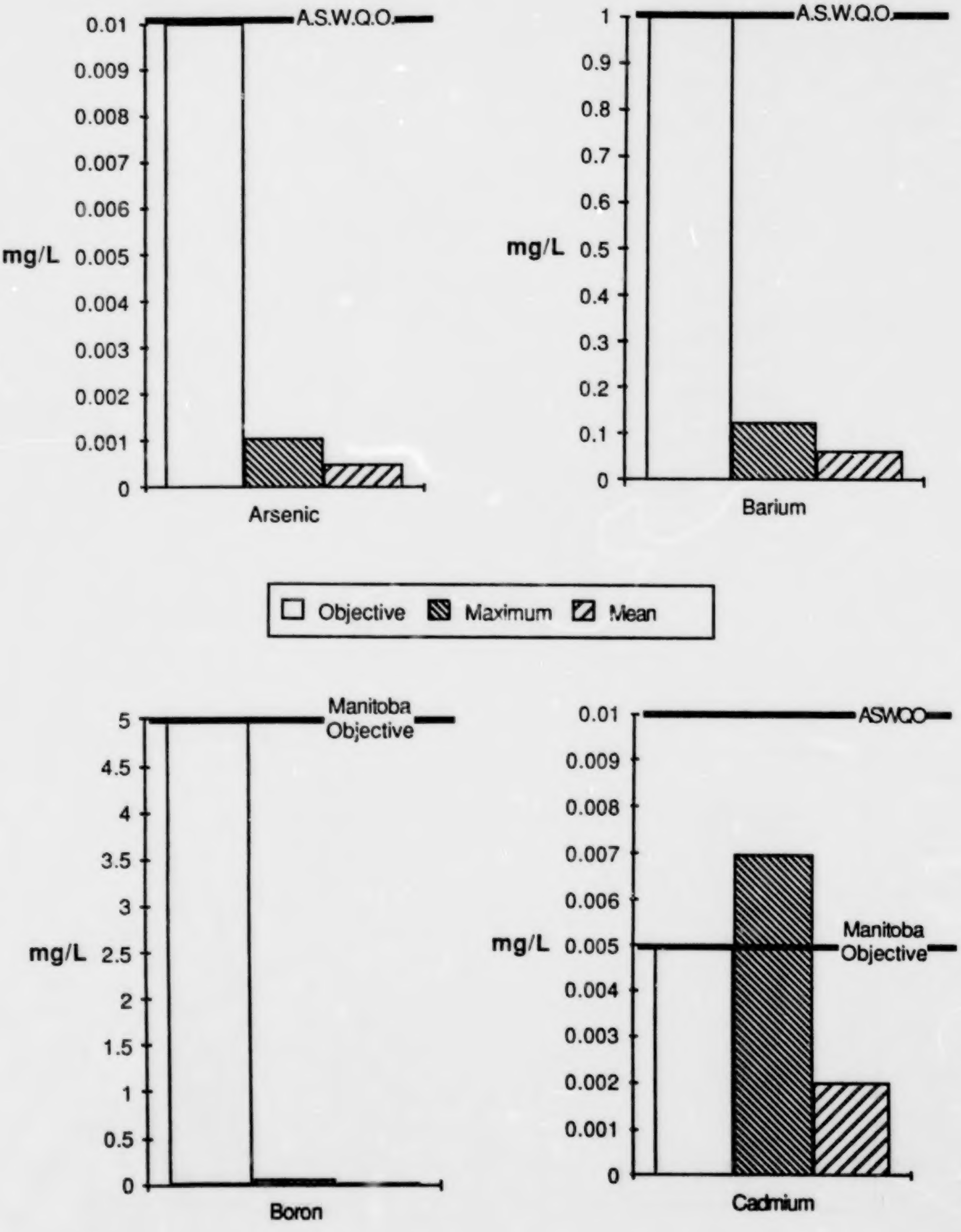


Fig. 2.5 to 2.8. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

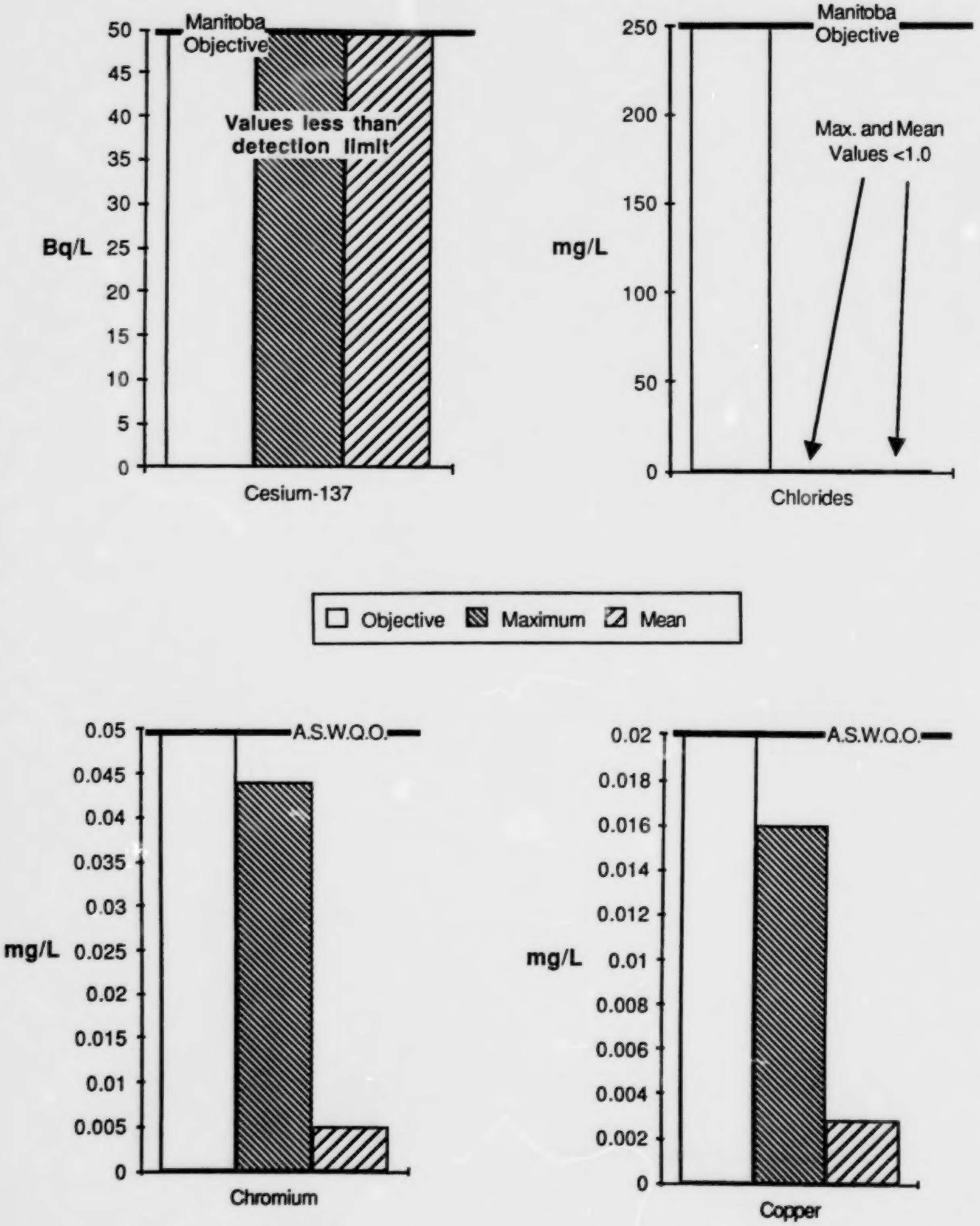


Fig. 2.9 to 2.12. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

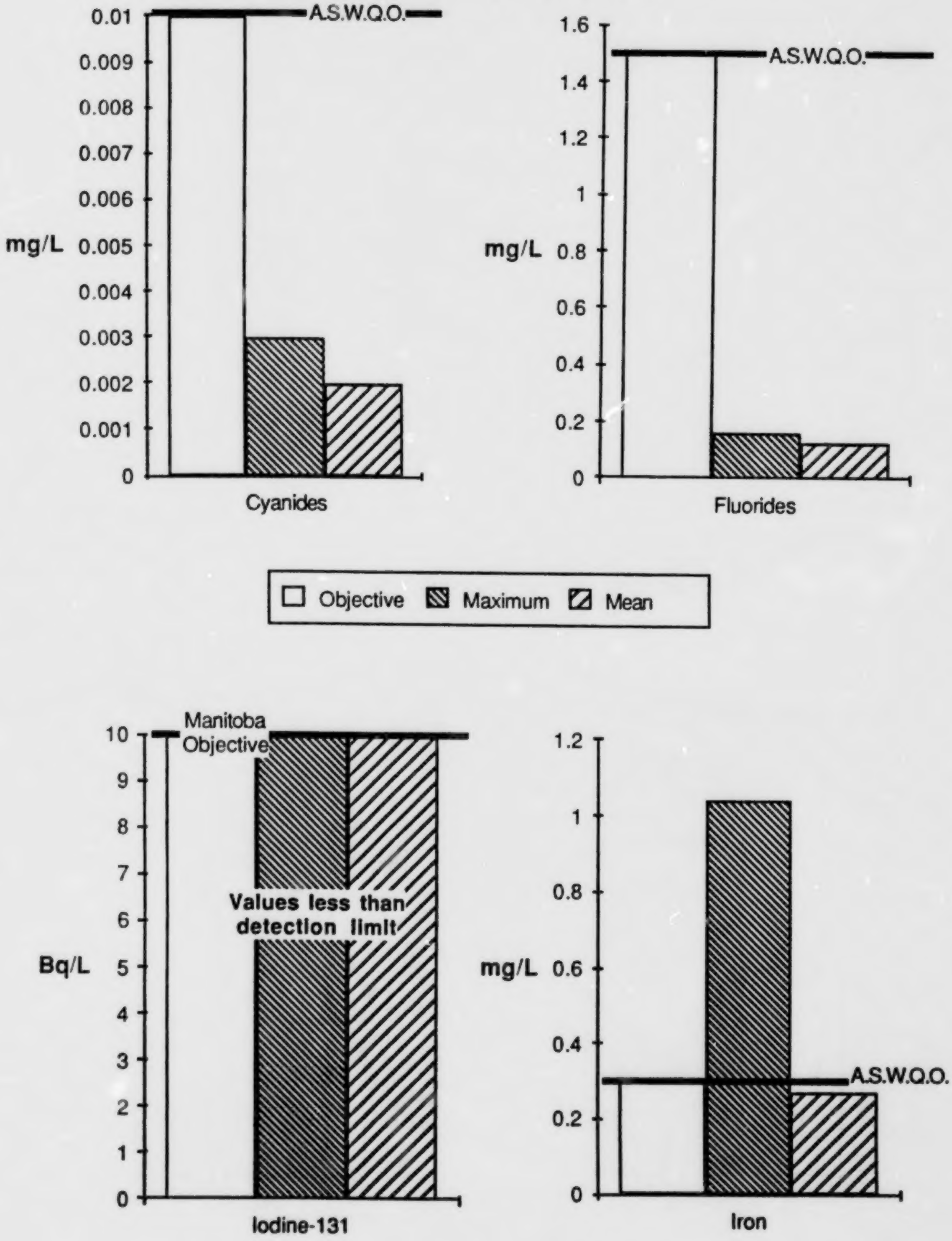


Fig. 2.13 to 2.16. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

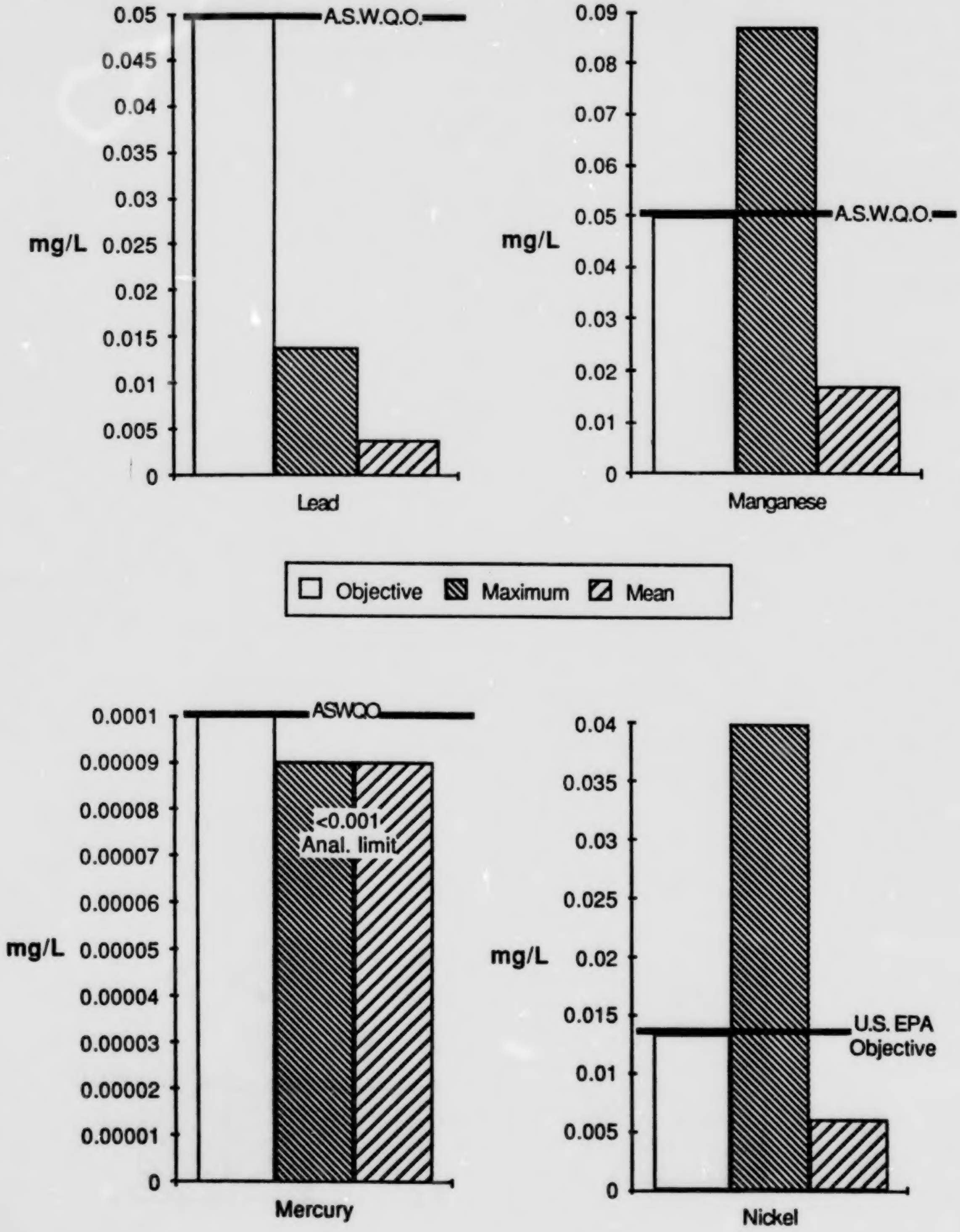


Fig. 2.17 to 2.20. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

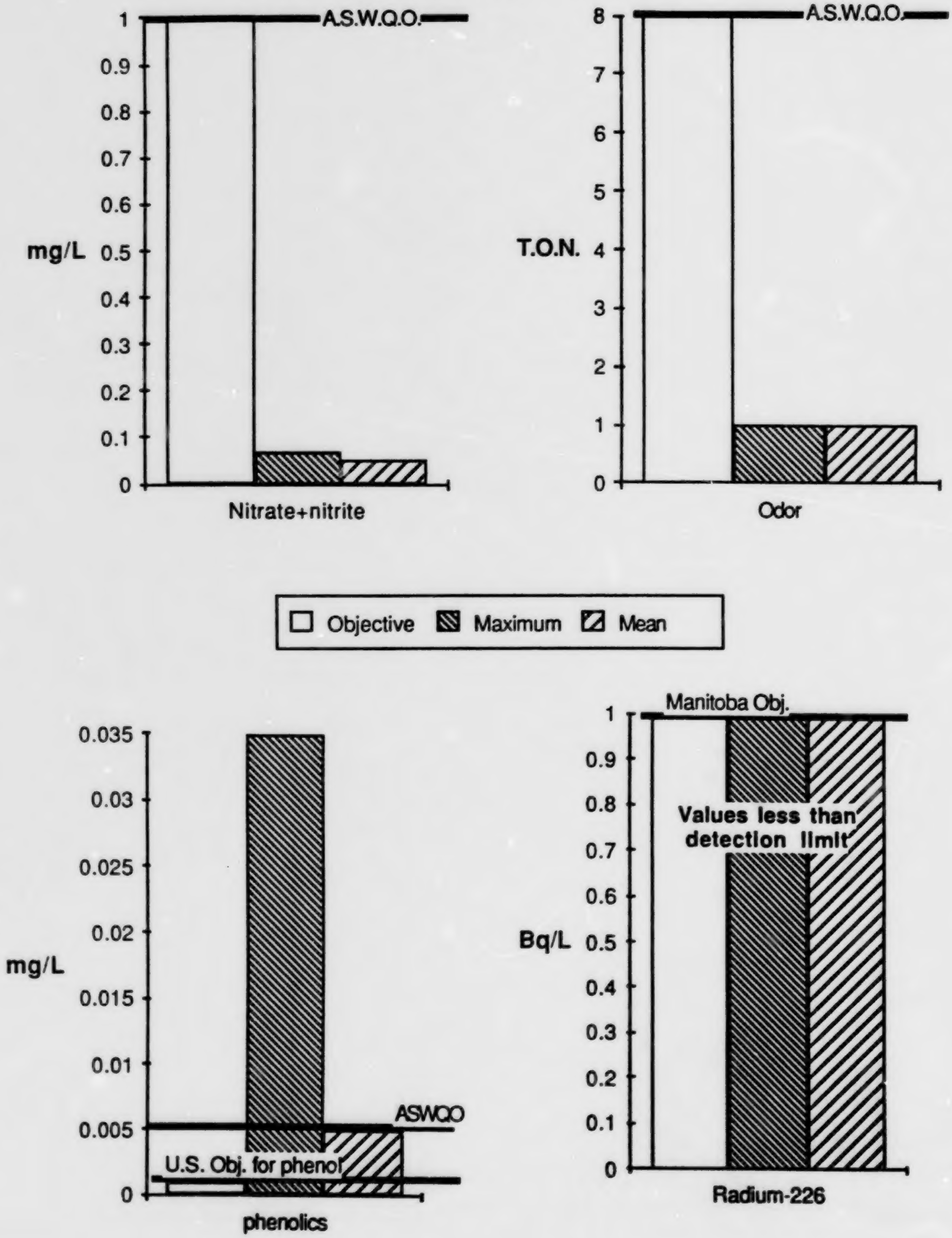


Fig. 2.21 to 2.24. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

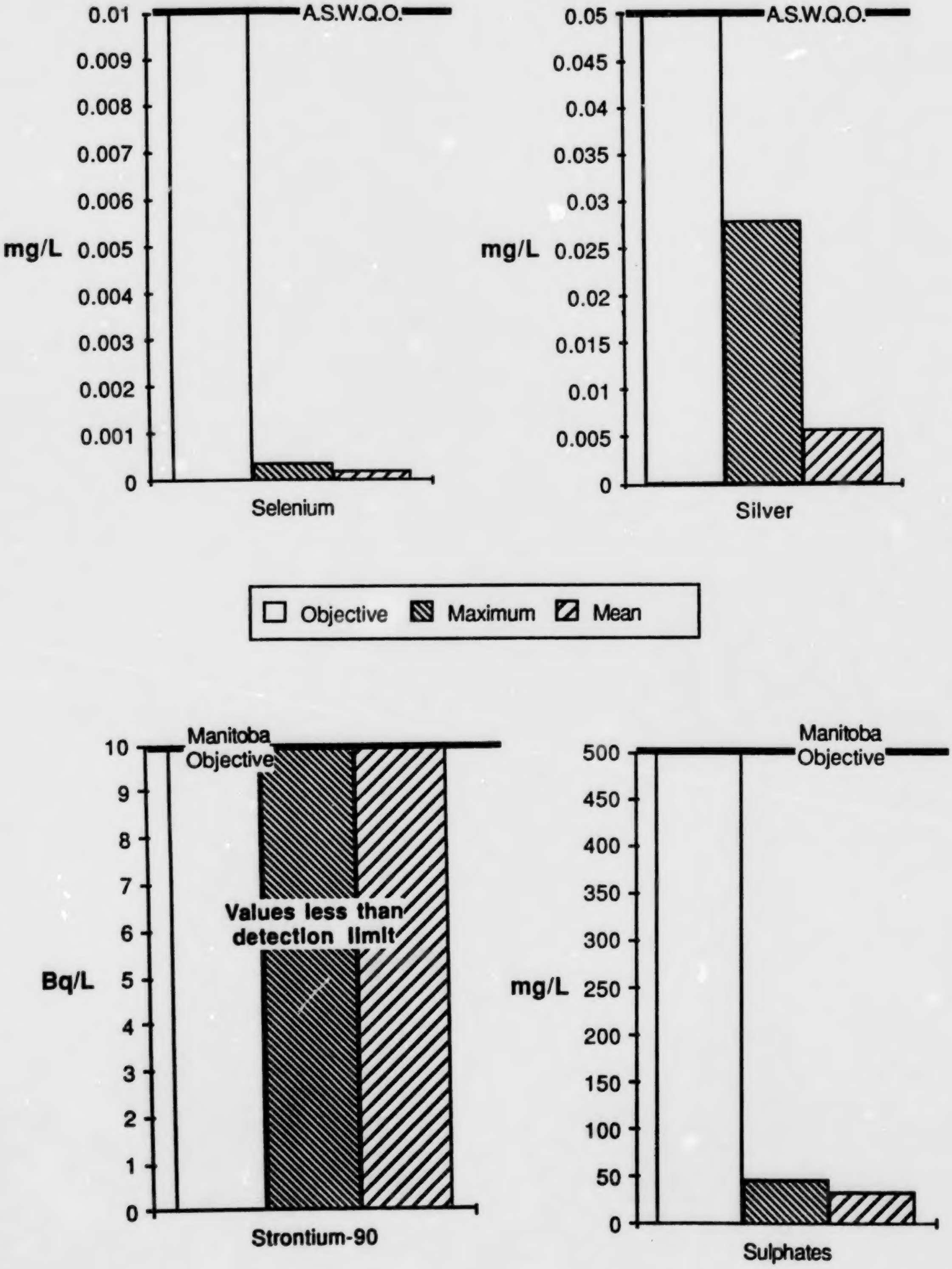


Fig. 2.25 to 2.28. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.

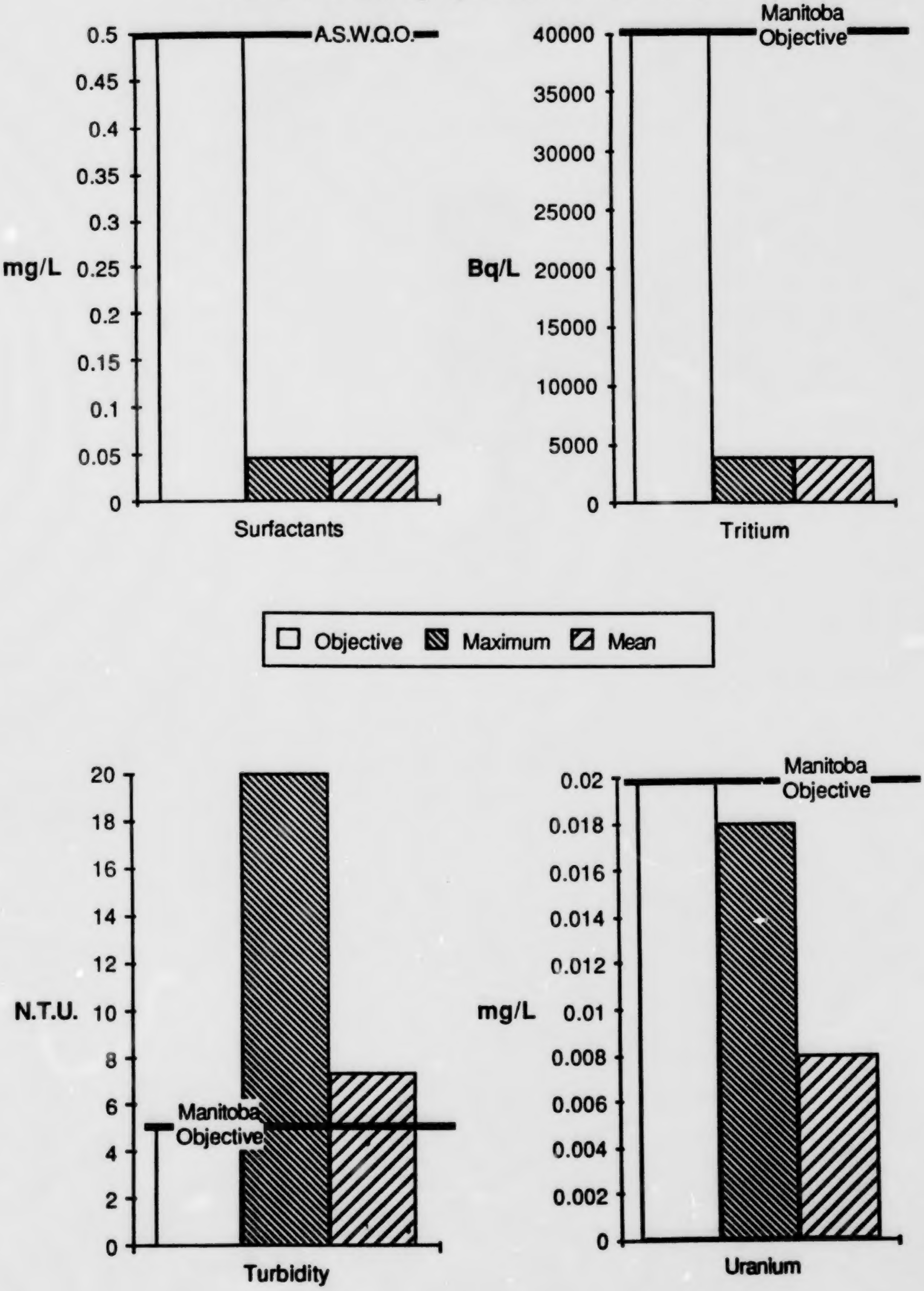
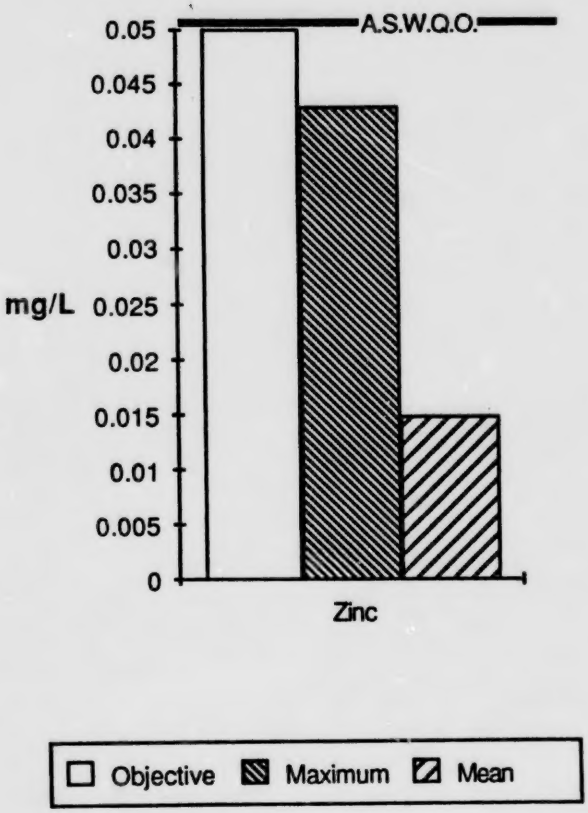


Fig. 2.29. Raw water quality for the North Saskatchewan River immediately upstream of Edmonton.



2.3 Trends in North Saskatchewan River Water Quality

The City of Edmonton and the Province of Alberta have grown rapidly since the Leduc oil discovery of 1947. The growth in population has been accompanied by major developments in the oil and gas industry. The growth in population and industrial activity combined with an improved awareness of environmental concerns leads some to expect that water pollution has increased at the same pace. That expectation requires careful examination.

Bouthillier[15] provided a personal account of over 35 years of water pollution assessment on the North Saskatchewan River. He recounts circumstances from the early 1950's of what would now rate as gross pollution. In one case, the newly opened Canadian Chemical Company plant was implicated for causing severe taste and odor problems with the North Battleford water supply because of discharges of untreated industrial waste to the North Saskatchewan River.

The City of Edmonton provided a major pollution load by discharging raw and partially treated sewage to the river. Winter dissolved oxygen levels were exhausted under ice cover during the 1950's for over 100 miles downstream. This condition would have made the river uninhabitable to all valued forms of aquatic life. By comparison, current winter values of dissolved oxygen downstream of Edmonton seldom drop below 70% of saturation and, correspondingly, pose no danger to aquatic life. Summer low values periodically approach levels of concern (5 mg/L of dissolved oxygen) likely because of vegetation growth in the river. Notwithstanding these existing water quality problems, there is substantial evidence that the overall water quality downstream of Edmonton has improved dramatically since the 1950's because of improved sewage treatment by Edmonton, reduced industrial waste discharges and flow regulation by the Brazeau and Big Horn Dams. Unfortunately, this knowledge does not provide much information on the status of the river upstream of Edmonton which is the concern for Edmonton's water supply. During the 1950's and 1960's the gross pollution conditions downstream of Edmonton prevented much attention from being directed towards upstream monitoring.

Reynoldson[16] reviewed North Saskatchewan River water quality data from 1970 to 1981. He considered routine water quality for 64 parameters at 4 sites: Devon, Waskatenau, Pakan and the Saskatchewan border. For evaluation of the Edmonton water supply, only the Devon site is relevant. Reynoldson grouped data into two categories: 1970-77 and 1977-81. Mean values were available in both categories for 55 of the parameters at the Devon site. Of these, only 5 showed higher mean values for the 77-81 period than for the 70-77 period. Comparisons of this type are extremely limited because of changes in analytical procedures and, even more important, variations in the times of sampling from year to year. The latter is critical because of the major seasonal fluctuations experienced of many parameters as the character of natural water input to the river changes.

The 5 parameters which showed higher mean values for the later period were total inorganic carbon, turbidity, silica, potassium and conductance.

The total inorganic carbon mean for 77-81 was 30 mg/L compared with 25 mg/L for 70-77. Results were based upon 28 samples for 70-77 and only 5 samples for 77-81. This does not provide a sound basis for the comparison of mean values. Furthermore, the higher mean values for the later time period were not supported by the bicarbonate data. Because bicarbonate would be the major source of inorganic carbon, the difference observed between the two time periods cannot be taken as significant.

The turbidity mean values were 15 for 77-81 versus 8 for 71-77. This difference might reflect an increase in turbidity but the value cannot be proven as significant relative to the range of natural seasonal fluctuations observed at this site. The maximum value at this site was 57 for the 70-77 period and 104 for the 77-81 period. Because turbidity values can change dramatically, even over the course of a day, more intense sampling at equivalent times of the year would be necessary to demonstrate a significant increase in this parameter. A check of the annual average raw water turbidities from 1980 to 1985 at the E.L. Smith plant, where at least daily turbidity analyses are conducted, provides no evidence of even a subtle increase in turbidity over the later time period.

Silica showed a mean value of 4.4 mg/L for 77-81 versus 3.8 mg/L for 70-77. The earlier period was based upon 13 samples versus 38 samples for the later period. This does not provide a reliable basis for judging the significance of a relatively small difference in mean values.

Potassium showed a mean value of 0.9 mg/L for 77-81 versus 0.8 mg/L for 70-77. Although the number of samples in each case is similar (47 for 70-77, 42 for 77-81), the small difference in mean values is unlikely to be significant given the range of values for each period up to 3.7 and 3.5 mg/L respectively.

Conductance showed a mean value of 327 usiemen/cm for 77-81 versus 317 usiemen/cm for 70-77. Again, the difference is small and this parameter, as an indication of total dissolved solids in the water, will be strongly influenced by variations in total river discharge. Such variations, both seasonally and annually will confound a subtle comparison. A total dissolved solids record was not available in Reynoldson's review for the 77-81 period to compare with the conductance values.

Overall, Reynoldson[16] concludes that there is no evidence of any deterioration in water quality over the 1970 to 1981 time period. The monitoring data he reviewed support that conclusion.

An additional perspective on possible trends in water quality was developed by reviewing data from provincial, federal and city sources dating from 1985 back to 1961.[14,17-23] This review consisted of plotting data for 13 parameters, focusing on those available which are related to the Canadian Drinking Water Guidelines for each of four monitoring sites: Whirlpool Point (near the river head waters), Rocky Mountain House, Devon and Edmonton near the 105 St. Bridge. These parameters were plotted individually, with annual median and maximum values, for every year of record available from the referenced sources. They are provided in Appendix A. Comparisons are somewhat confounded by inconsistent records at the various sites and differences in analytical techniques from year to year. In particular, metals values have changed among dissolved, total and extractable categories. Notwithstanding the limitations imposed by the varying factors, there is no evidence in the available data to suggest any trend toward deteriorating water quality upstream of Edmonton.

2.4 Comparison with Other Raw Water Sources

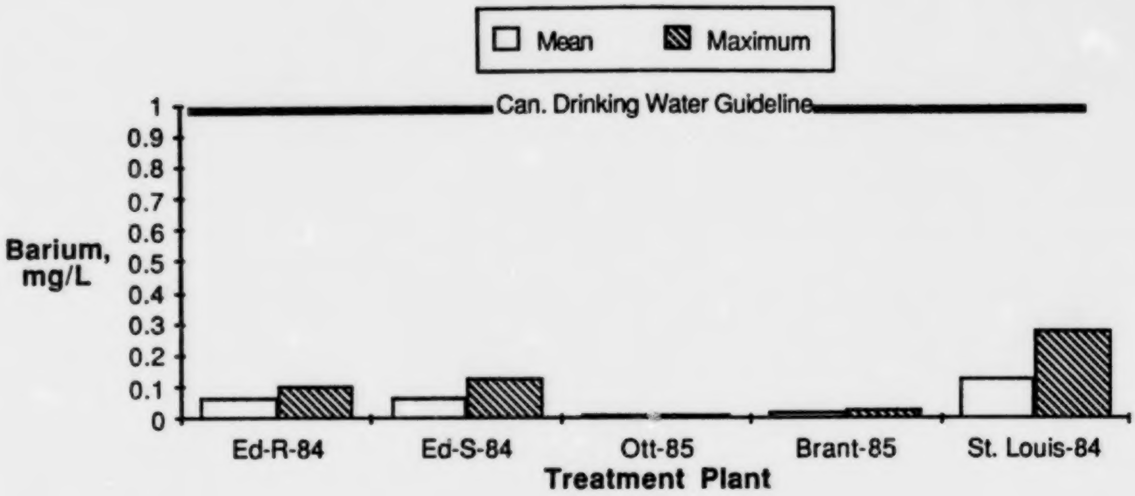
2.4.1 Sources of Data for Comparison

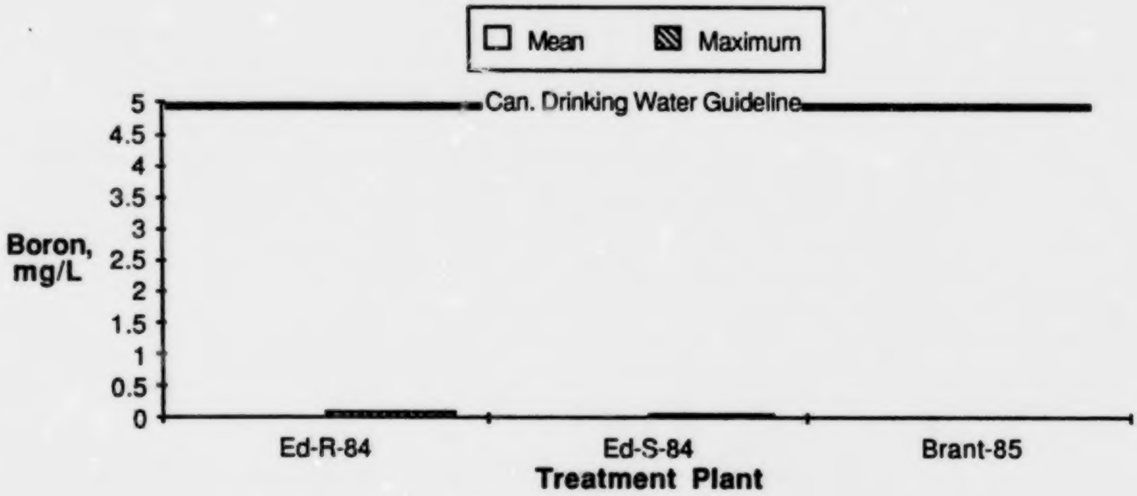
In addition to the status of the North Saskatchewan River relative to surface water quality objectives it is informative to compare this source with those used by other cities in Canada, the United States and Europe. For comparison, plants were selected by the treatment experts who compared the Edmonton plants with practice elsewhere.

Mr. P. Foley provided raw water data for water treatment plants at Ottawa[24] and at Brantford[25]. Dr.P.Huck provided raw water data for plants at Zurich, Switzerland[26] and at Langenau, Germany[27]. Dr.V.Snoeyink provided raw water data for the St.Louis County plants[28] in the United States. Detailed descriptions of those facilities and their respective circumstances are provided in the reports by the respective treatment experts in this Volume. The plants were selected because they experienced some similarities with Edmonton's situation. Their raw water sources certainly do not reflect the worst which are commonly encountered by large urban centers.

Data was extracted for Edmonton plants from the 1984 Water Treatment Plants Annual Report[30]. Annual mean and maximum values are compared for the parameters specified as health-related by the Canadian Drinking Water Guidelines[10] with corresponding data, where available, for the other raw water sources. These are plotted in Figures 3.1 to 3.12.

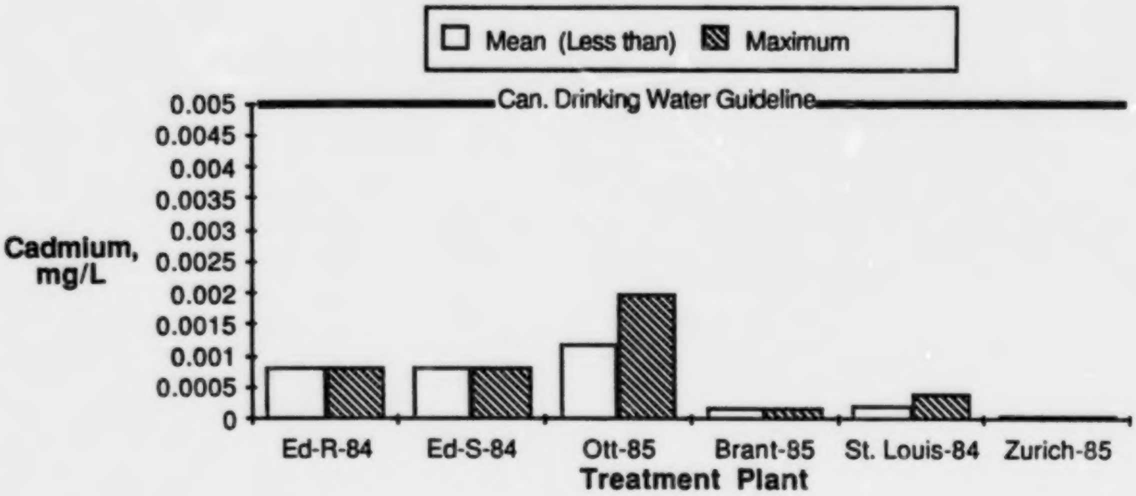
Fig. 3.1 to Fig. 3.2. Edmonton's raw water quality compared with that of other cities (both national and international).

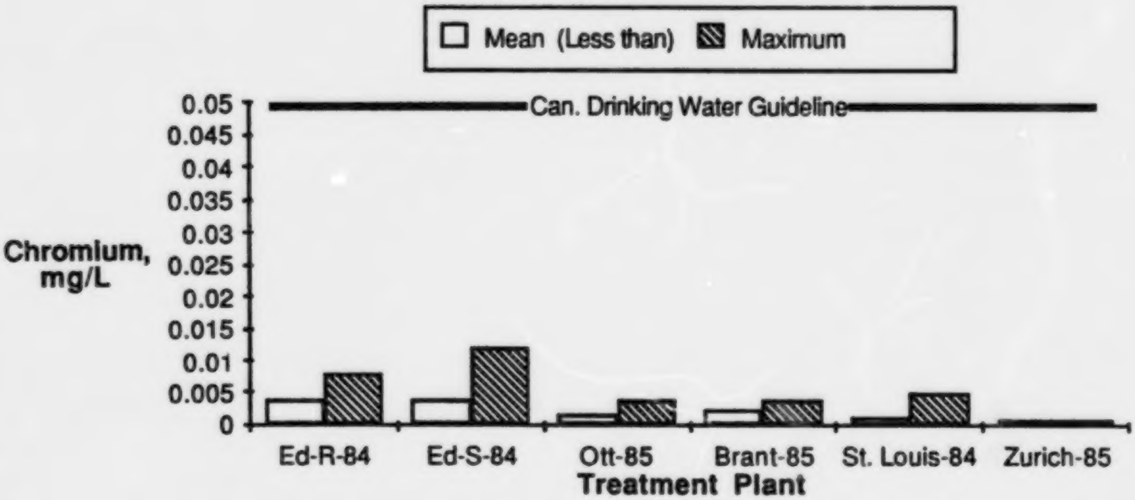




Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

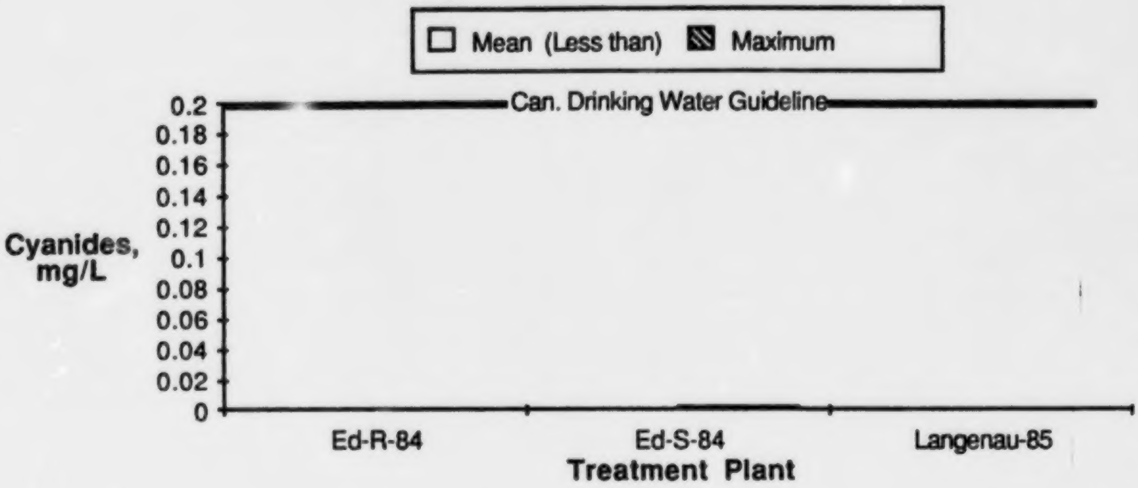
Fig. 3.3 to Fig. 3.4. Edmonton's raw water quality compared with that of other cities (both national and international).

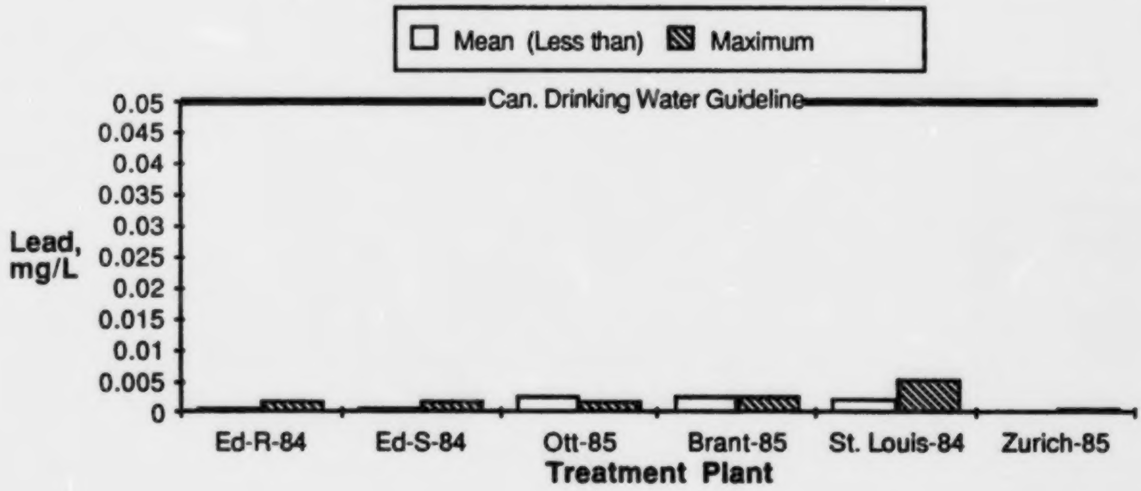




Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

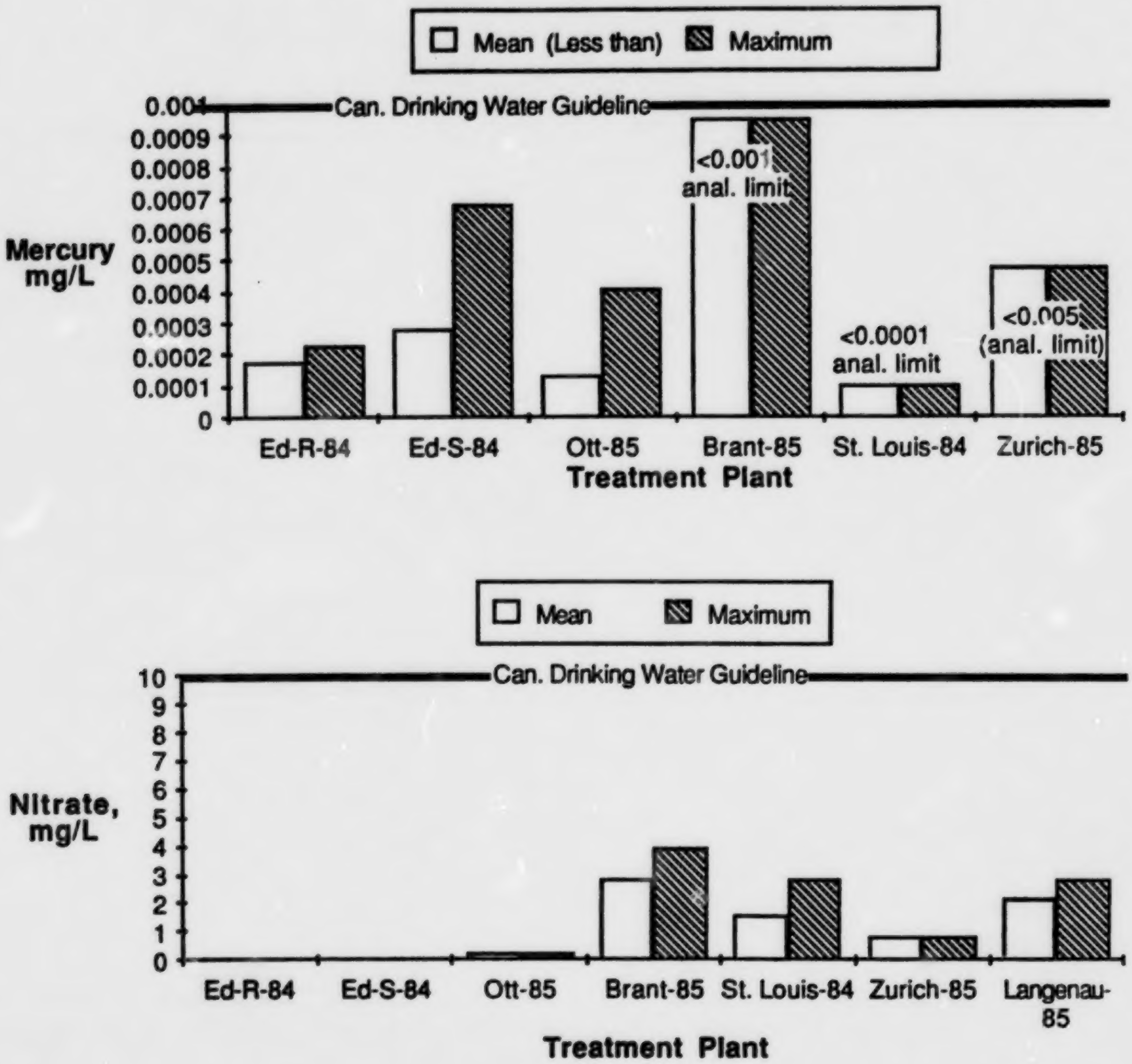
Fig. 3.5 to Fig. 3.6. Edmonton's raw water quality compared with that of other cities (both national and international).





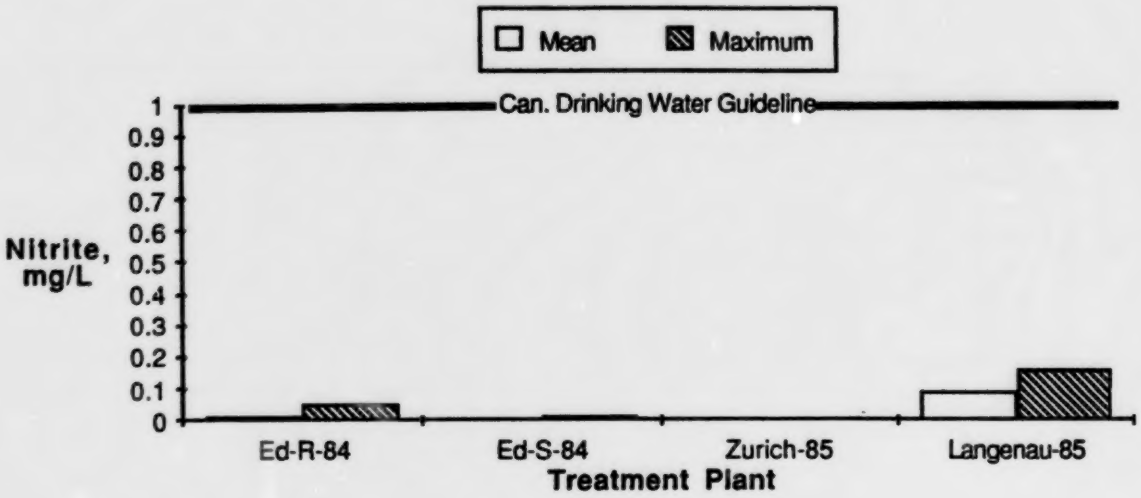
Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

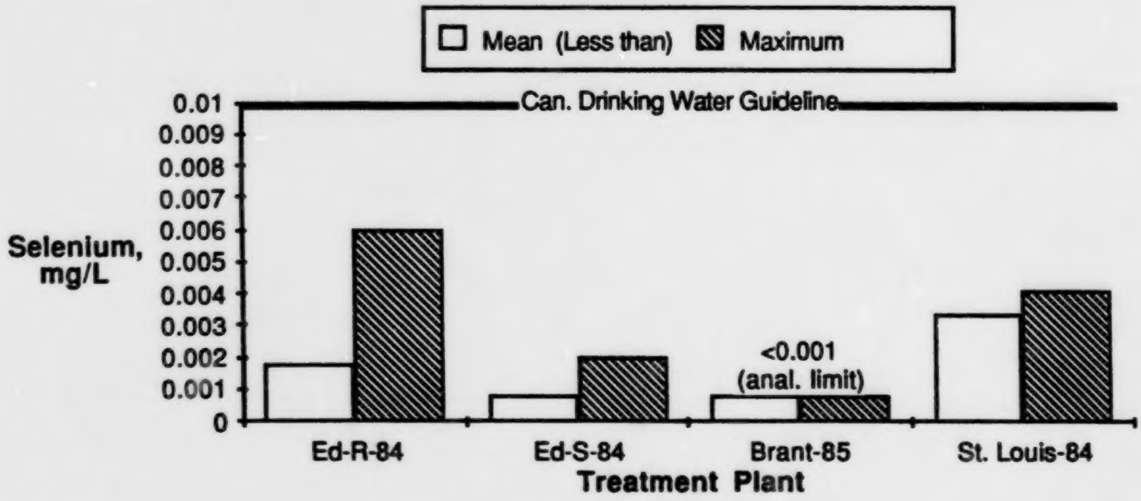
Fig. 3.7 to Fig. 3.8. Edmonton's raw water quality compared with that of other cities (both national and international).



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

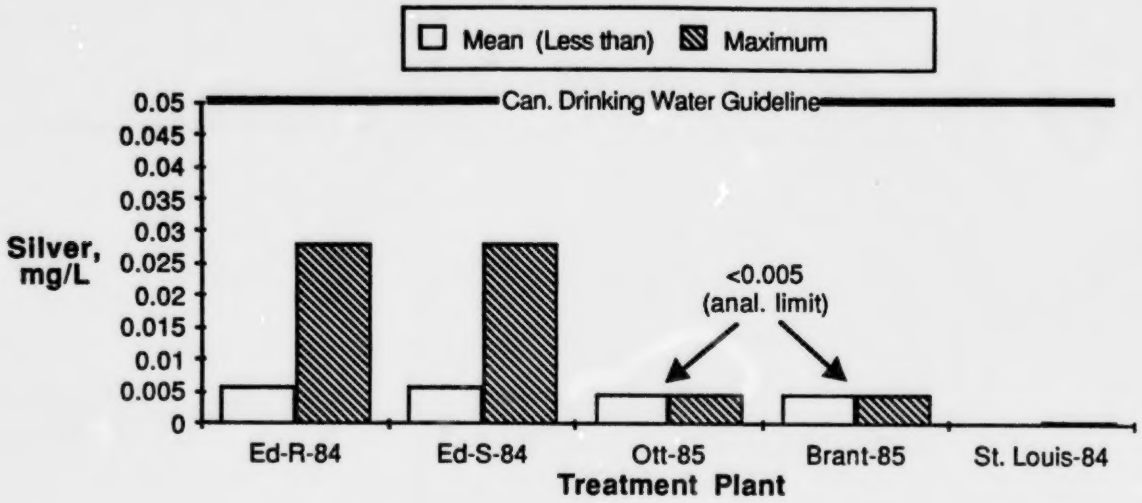
Fig. 3.9 to Fig. 3.10. Edmonton's raw water quality compared with that of other cities (both national and international).

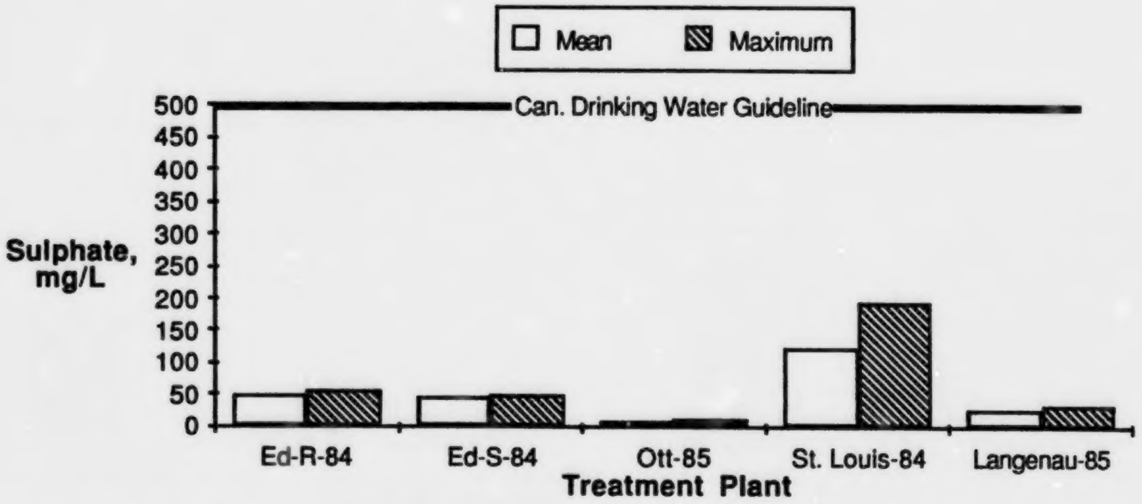




Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 3.11 to Fig. 3.12. Edmonton's raw water quality compared with that of other cities (both national and international).





Ed-R-84 = Edmonton Rossdale, 1984
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Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

2.4.2 Discussion of Comparisons

2.4.2.1 Arsenic. Both annual mean and maximum arsenic values for raw water from all sources are well below the levels specified as safe in treated drinking water let alone raw water.

2.4.2.2 Barium. Both annual mean and maximum barium values for raw water from all sources are well below the levels specified as safe in treated drinking water let alone raw water.

2.4.2.3 Boron. Both annual mean and maximum boron values for raw water from all sources are well below the levels specified as safe in treated drinking water let alone raw water.

2.4.2.4 Cadmium. Most mean and maximum values are below the analytical detection limit and all are certainly well below the levels specified as safe in drinking water let alone raw water.

2.4.2.5 Chromium. Most mean values are below the analytical detection limit. The mean values for the two Edmonton plants are less than 0.004 mg/L because they include values below the detection limit in the calculation. Although maximum values for Edmonton appear higher than the comparable raw water sources the values are well below the levels specified as safe in drinking water let alone raw water.

2.4.2.6 Cyanides. The only plant other than the Edmonton plants which reported cyanides for raw water was the Langenau plant in Germany. In the case of the Edmonton plants values were generally not detectable and maximum values were marginally above the analytical detection limit. All values were well below the levels specified as safe in drinking water let alone raw water.

2.4.2.7 Lead. Mean and maximum values for the North Saskatchewan River were generally less than those reported for the Ottawa River and the Grand River (Brantford). In all cases the values are well below the levels specified as safe in drinking water let alone raw water.

2.4.2.8 Mercury. The values reported for mercury appear somewhat higher but this is only apparent on a relative basis because the guideline for mercury is much lower than for other substances. Most values were actually near their analytical detection limit. This is particularly true for Brantford which reported mean and maximum values as less than 0.001 mg/L which is the guideline value[10]. One value at Smith was reported at 0.0007 mg/L which is close to the guideline value. Although the guidelines are based upon long term average consumption, the occasional high mercury value suggests a need for confirmation that the values are indeed valid and not an artifact of sampling or analysis. No equivalent values upstream of Edmonton were reported by Alberta Environment[14] or the river monitoring program for the snow dumps.[23,24]

2.4.2.9 Nitrate. Mean and maximum values for Edmonton are barely detectable and reflect the limited upstream activity on the North Saskatchewan River. By comparison, the values for Brantford, Langenau and St. Louis County reflect the influence of upstream discharges. In this comparison, the North Saskatchewan River is clearly superior to most of the other raw water sources used for comparison.

2.4.2.10 Nitrite. Values for nitrite are consistently below levels specified as safe for drinking water.

2.4.2.11 Selenium. Brantford was the only other source which reported values for selenium. In most cases, values were below the analytical detection limit except for a maximum value reported at Rossdale which is more than 50% of the maximum acceptable concentration for selenium in treated drinking water (as opposed the raw water under discussion). Alberta Environment monitoring data for the North Saskatchewan River upstream of Edmonton[14] have routinely shown selenium at below a detection limit of 0.0002 mg/L. As was recommended for mercury, the City of Edmonton should exercise additional quality control in the sampling and analysis for selenium.

2.4.2.12 Silver. Raw water values for silver were generally reported as below the detection limit for all sources but maximum values were reported for both Edmonton plants as <0.03 mg/L. The meaning for that designation is not clear. Other monitoring programs on the North Saskatchewan River do not report silver so there is no basis for comparison. The comments on sampling and analytical quality control made for selenium and mercury also apply here.

2.4.2.13 Sulphate. Raw water values for sulphate from the North Saskatchewan River are higher than observed at Ottawa and Langenau but are less than observed at St. Louis county. In all cases, the values are well below levels considered a concern for drinking water consumption.

2.4.2.14 Pesticides. Because data on pesticides in raw water were not obtained except from Ottawa, charts were not prepared for comparison. As noted in section 2.1.2 the only pesticides to be detected in the North Saskatchewan River upstream of Edmonton were alpha-BHC, gamma-BHC, Lindane and 2,4 D. All were found at extremely low levels, namely, 1/4000 (Lindane) to 1/10000 (2,4 D) of the levels considered safe in treated drinking water according to the Canadian Drinking Water Guidelines.[10] Ottawa reported analyses for chlorinated pesticides and herbicides in the Ottawa River. They only found alpha-BHC at levels up to 0.000007 mg/L which is only marginally higher than the maximum value of 0.000005 mg/L found for the North Saskatchewan River. The BHC compounds do not have a specific guideline level for drinking water.

2.2.4.15 Summary. Overall the North Saskatchewan river compares favorably as a raw water source with the locations chosen for comparison and described for treatment practice in the expert reports. All sources compare favourably with the Canadian Drinking Water Guidelines.[10] The raw water sources chosen for comparison range from fair to very good.The North Saskatchewan River would rate as a good overall raw water source on a comparative basis.

There were three cases for the North Saskatchewan River, mercury, selenium and silver where maximum values reported for the raw water were more than 50% of the Canadian Drinking Water Guideline value for treated drinking water (rather than raw river water). Because the higher values are not generally supported by other monitoring being done on the North Saskatchewan River, the City of Edmonton should direct specific quality control efforts at the sampling and analysis for these parameters.

2.5 Point Source Discharges Upstream of Edmonton

There is very little development, either municipal or industrial located on the North Saskatchewan River upstream of Edmonton.

The first direct point source wastewater discharge to the North Saskatchewan River, travelling downstream from the headwaters, is from the municipal sewage lagoons for the town of Rocky Mountain House. These consist of 4 short term detention lagoons and 2 long term detention lagoons which biologically treat the municipal sewage from a population of approximately 5000. They are licensed for discharge only during spring high flow and again in the fall after downstream farming operations have ceased. This discharge occurs to the right bank of the North Saskatchewan River approximately 300 km upstream from Edmonton. Rocky Mountain House is in the process of upgrading to aerated lagoons which will discharge on a continous basis.

The second direct point source discharge occurs from Drayton Valley. This system involves aerated lagoon treatment of municipal and local industrial waste followed by chlorine disinfection. This system provides continuous discharge to the left bank of the North Saskatchewan River via a small unnamed creek. The discharge occurs approximately 200 km upstream from Edmonton.

The third direct input arises from the TransAlta Utilities Sundance power plant on Lake Wabamun. This plant draws cooling water from the North Saskatchewan and occasionally returns flow from its cooling pond to control dissolved solids buildup. Approximately half of the water withdrawn from the river is returned as blowdown which suggests at least a two-fold increase in dissolved solids for the return flow. This cooling blowdown is controlled for suspended matter, total iron, pH and oil and grease. The latter is limited to a maximum concentration of 5 mg/L or 275 kg/day. This discharge occurs approximately 100 km upstream of Edmonton.

The final direct point source discharge is from the town of Devon with a population of about 4000. Municipal sewage is treated by primary clarification, secondary (biological) treatment in a modern rotating biological contactor. The effluent is disinfected with chlorine before discharge to the right bank of the North Saskatchewan River. This discharge occurs approximately 20 km upstream of the E.L. Smith intake which is located in the center of the river channel.

In addition to these fixed discharge activities, some gravel mining operations were observed on the river during a helicopter trip up to the Brazeau Reservoir on May 12, 1986. Generally, such activities should cause effects limited to turbidity disruptions of water quality which can be controlled, onsite, by proper settling facilities. However, there is a potential for chronic hydrocarbon contamination from poorly maintained equipment and acute contamination from spills. These possibilities need to be assessed in more detail by conducting sampling surveys and inventories at some of the existing operations.

Indirect point source discharges, which reach the North Saskatchewan River by various tributaries, arise from several small municipal sources and from the Transalta Utilities Wabamun power plant. The municipal sources are all seasonal discharge lagoon systems. These include: Alder Flats, Violet Grove, Rocky Rapids, Tomahawk, Breton, Winfield, Warburg, Sunnybrook, Thorsby, Buford, Calmar, Nisku, Edmonton International Airport and Kavanagh. The Nisku and airport facilities are being connected to the South East Regional Trunk Sewer and will soon be directed to the regional sewage treatment plant downstream of Edmonton.

The Wabamun power plant draws cooling water from Lake Wabamun and discharges it directly back to the lake on a once-through basis. The plant also decants wastewater from its ash storage lagoon and from the east mine drainage pond to the lake. These are controlled for suspended matter, total iron, total phosphorus and pH, while the ash lagoon is also limited for oil and grease (average daily discharge less than 30 kg, maximum less than 125 kg). Lake Wabamun, in turn, discharges to the left bank of the North Saskatchewan River via Wabamun Creek, approximately 100 km upstream from Edmonton.

2.6 Runoff Discharges to the North Saskatchewan River

2.6.1 Urban Contributions

__2.6.1.1 Storm Sewer Contributions.__ Within the City of Edmonton there is a mixture of drainage systems with an older combined sewer system in the central core and a separated sewer system in areas built since about 1950. The combined sewer system, which will overflow a mixture of surface runoff and raw sewage to the river during storm events, serves approximately 19% of the developed area of the city.[29] There are a total of 7 overflows for this combined sewer system located upstream from the Rossdale water intake. There are another 78 separated storm sewer outfalls located upstream of the Rossdale water intake. Of this total, at least 22 are known to carry some quantities of sanitary sewage. The 7 combined sewer overflows have been identified as a high priority concern and work is underway to eliminate them by 1987.

Apart from the known sanitary sewer overflow connections to the storm sewer system, there are also illegal or unintentional cross-connections which can discharge sanitary sewage to storm sewers. The concerns associated with untreated sanitary sewage discharge upstream from a water intake relate mainly to the associated risk of pathogenic microorganisms challenging the drinking water treatment plant. These concerns have been addressed in the expert reports by Drs. Logsdon, Payment, Smith and Tobin and they will not be reiterated here.

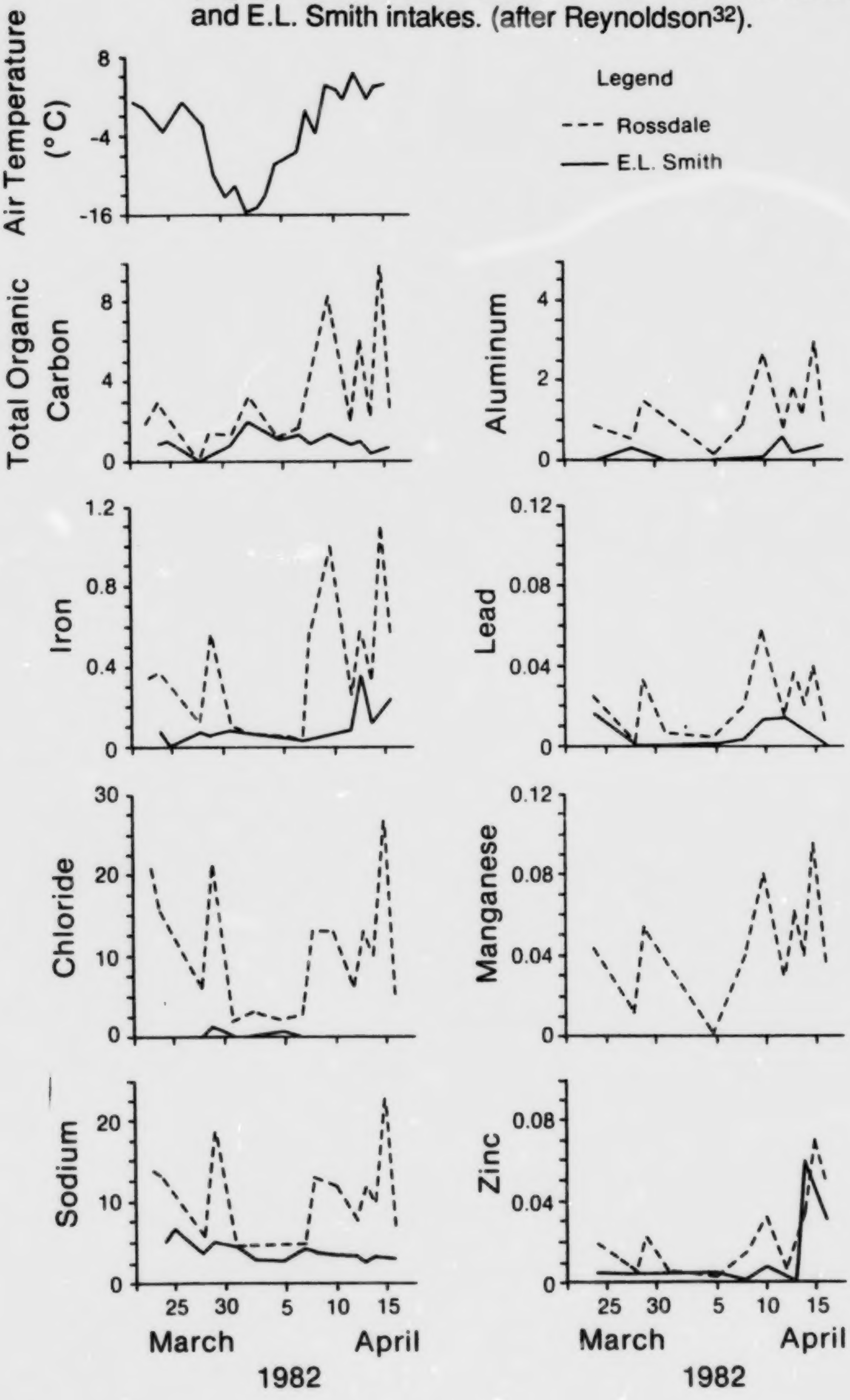
Storm runoff, without sanitary sewage, is still a source of contamination which may cause problems for a drinking water supply. More specific to Edmonton, Reynoldson and Livingston conducted a survey[32] during March and April 1982 to determine the effect of urban runoff on the quality of the North Saskatchewan River upstream from the Rossdale intake. They concluded that storm runoff was responsible for significantly higher values of several metals (lead, copper, iron, manganese and zinc) and chlorides at the Rossdale plant compared with the Smith plant. However, since average values for these metals met the ASWQO and maximum values did not dramatically exceed the ASWQO, the differences shown between Rossdale and Smith did not represent a compelling degradation in water quality. They did, however, estimate total loadings to the river attributable to storm sewers upstream of Rossdale. These included values such as lead ranging from 15 to 73 kg/day and dissolved organic carbon ranging from 3.7 to 5.2 tonnes/day.

Limited results were also reported for trace organic analyses from sewer samples. These suggested a total level of 850 ug/L (ppb) of specific organics in a Groat sewer sample with the major components being identified as alkylated benzenes, alkylated naphthalenes and branched alkanes and alkenes. Such materials are characteristic of gasoline, kerosine and fuel oils. Given threshold odour levels[31] of only 1.6 ug/L (ppb) for ethylbenzene and 0.8 ug/L (ppb) for no.2 fuel oil, the significance of these compounds being discharged immediately upstream from the Rossdale intake should be apparent.

This report also plotted daily values for several parameters at the Rossdale and Smith plants. Inspection of these data, reproduced in Figure 4, indicate that the Rossdale plant experienced widely fluctuating water quality relative to the E.L. Smith plant. Perhaps most informative of these parameters is the total carbon (presumed by the values reported to actually be total organic carbon) which would be indicative of the organic loading on the two plants. The considerable fluctuations experienced at the Rossdale plant, relative to the E.L.Smith plant, would be sufficient to cause significant challenges to the conventional treatment process employed.

In 1983, further monitoring by Reynoldson and Livingstone[33] included samples from the Groat sewer which were analyzed by gas chromatography / mass spectrometry (GC/MS). These again documented concentrations in the 10 to 500 ug/L (ppb) range for a series of alkylated benzenes as well as comparable concentrations of tetrachloroethylene (a common dry cleaning and industrial solvent and a U.S.E.P.A. classified "probable human carcinogen"). Unfortunately, despite the major sampling and analytical efforts in 1982 and 1983, the extent of the impact of the urban runoff on the Rossdale intake was not clearly demonstrated by focusing the reporting on the types of contaminants which would cause the taste and odor complaints which had been common to Rossdale

Fig. 4. Influence of urban runoff on selected chemical parameters (mg/L) from the North Saskatchewan River at Rossdale and E.L. Smith intakes. (after Reynoldson[32]).



water during spring runoff. Likewise, health concerns with trace organics were not highlighted in reporting of these surveys.

Edmonton Water and Sanitation took an initiative with a sampling program involving 19 samples of raw and treated water from Rossdale and Smith. These were collected on March 29, April 15 and April 23, 1982. These were subjected to GC/MS analyses by Kinetic Environmental Research Laboratories.[34] Results indicated the presence of detectable hydrocarbons in all raw water samples from both Smith and Rossdale. The lab reports distinguish hydrocarbons from aromatics, a peculiar convention because aromatics are hydrocarbons. Presumably, the first designation is intended to mean aliphatic hydrocarbons. Chorinated hydrocarbons were also commonly identified but doubts were raised about the purity of the solvents used in the analyses and the reagent blanks were significantly contaminated. Chlorinated phenols were also identified in several raw samples. However, identifications, in general, were based on computer library matching. This approach is unreliable and inadequate for confirmed identification, particularly for complex samples where chromatographic resolution may be poor. Dr. Coutts, based upon his interpretation of the mass spectra obtained (see Coutts report, this Volume), concluded that identifications of compounds other than hydrocarbons were generally wrong.

Overall, this survey was a commendable attempt by the water laboratory to understand the nature of the organic contamination problem. However, the results provided are sufficiently suspect that they provided an inadequate basis for problem resolution. The survey did apparently result in a summary report by lab staff[35] which proposed to follow this study with more frequent GC analyses of raw and treated water and: "To scan for purgeable compounds using extraction as well as purge and trap methods." The latter is a contradiction, in that extraction will miss the more volatile purgeables. In any case, extensive use of purge and trap GC has been implemented, but extraction procedures have not been routinely adopted. Likewise, proposals for onsite pesticide and chlorinated phenols analysis, using electron capture and nitrogen/phosphorus flame ionization detection, have not been implemented.

Hydrocarbon impact on the Rossdale intake was clearly demonstrated by a raw water sample taken at the plant September 28, 1984. When analyzed by Enviro-test Laboratories[36] this sample indicated a total of approximately 1500 ug/L (ppb) of a complex hydrocarbon mixture. The chromatogram was strongly characteristic of that type of mixture and concentrations were more than adequate to assure confirmed mass spectral identification. Given that oil slicks had been observed from time to time within the process at Rossdale[37], periodic oil contamination of the Rossdale intake seems to have been well known.

A major city study[38], covering the period from 1980 to 1984, addressed the characteristics of storm sewer discharges to the river. This program involved collection of 707 composite samples of storm sewage with a total of 10,206 analyses performed. The report found that storm runoff was not a major pollution source (using the ASWQO as a guide) for arsenic, barium, boron, fluoride, selenium and silver. Cadmium, cyanide, mercury, iron, manganese and zinc were found to regularly exceed ASWQO and were judged to be not amenable to reduction by point source control. Intermittent high values exceeding ASWQO were observed for chromium, lead copper, phenolics and sulphide. These were judged to be amenable to point source control. Although this report documents the poor water quality frequently associated with storm sewer discharges to the North Saskatchewan River it does not address the issue of organics, particularly hydrocarbons, carried by storm runoff.

2.6.1.2 Snow Dumps. The City of Edmonton has made use of direct dumping of snow collected from winter street clearing operations onto the ice-covered North Saskatchewan River.

This practice has been subject to an annual license under the Clean Water Act since 1973. Those annual licenses required the City to undertake monitoring programs to assess the quality of the material being placed in the river and its possible impact on river water quality. This program has created a very large data base which cannot be reviewed, in detail, for the purposes at hand. However, some highlights will serve to illustrate the potential significance of Edmonton's snow dumping practice on raw water quality at the Rossdale intake.

Early results from this program[39] are among the most interesting. Samples reported for sites at Dawson Bridge and at 122 St and 124 Ave (not a direct river dump, it would drain into storm sewers) included threshold odor numbers (TON) which were 512 and 2050 respectively. The odor character was described as hydrocarbon. The TON is a measure how much dilution with odor free water is necessary to bring a sample to a non-detectable odor.

Dilutions much higher than 500 to 2000 will ultimately be provided for snow melting from snow dumps, once completely mixed in the total river flow. However, that is not the nature of the mixing and dilution process in the river (see

Smith report, this Volume). The melting and dilution processes for materials such as hydrocarbons, which will likely not be uniformly distributed in the snow mass, allows for occasional slugs of high concentration material to pass through. Such slugs could, in turn, be intermittently drawn into the Rossdale intake. This possibility was particularly relevant when a snow dump site was located immediately upstream of Groat Bridge.

In 1979-80, higher values were encountered at the Groat site for the oil and grease parameter. As noted in section 2.2.1, this parameter has serious flaws for river monitoring, primarily because of insensitivity at the low concentrations normally encountered in rivers and the likelihood of interference from biological materials in those circumstances. The method is not ideal for concentrated pollution sources because of its non-specificity. However, it is at least a relevant parameter when conducted on materials where the most likely source of solvent extractable material will be hydrocarbons. The oil and grease value reported for the Groat Road site in 1979-80 was 530 mg/L. This value was significantly higher than obtained at this site in previous years. The report[38] explained this as: "High Oil and Grease result for 1979-80 sampling season was a result of improved sampling technique." That high value, when used with an estimate of total snow mass which was used for estimating the mass of individual metals in the dump, provides an estimate of 4.5 tonnes of "oil and grease" type material in the Groat dump. Confidence in the accuracy of this estimate would have to be rated as very low. However, the potential of substantial hydrocarbon material being present in the Groat snow dump was certainly raised by these results. Subsequent years sampling for oil and grease produced much lower values than 1979-80 and earlier.

No comment was offered in subsequent reports to explain the sudden decrease in observed oil and grease. The range of results for oil and grease reported over the years is summarized in Table 2. River monitoring for oil and grease, since 1982, upstream and downstream from snowdump sites, failed to show any influence from the dumps.

Analyses of eight snow dump core samples taken by Alberta Environment in 1982 showed oil and grease values to be less than 10 mg/L and trace hydrocarbons (method not specified) at less than 0.1 mg/L. These findings leave the actual overall contribution of snow dumps to intake water quality at Rossdale a moot point. However, there is little doubt that when the Groat site was used there was substantial likelihood for at least intermittent hydrocarbon contributions to the Rossdale intake. The nature of the monitoring data available do not allow determination of how significant these contributions may have been. Relocation of the Groat snow dump to avoid impact on the Rossdale intake is a simple, but necessary expedient which should be maintained.

As with the storm sewers, considerable data has been collected on the contribution of metals from snow dumps to

Table 2 Oil and Grease for Snow Dump Monitoring[39]

Year	Snow Dump Site (oil and grease measure)	
1973	Dawson (826 mg/L)	122 St. & 124 Ave. (430 mg/L)
1974	Groat (107 mg/L)[1]	105 St. & 45 Ave. (171 mg/L)[2]
1975	Buena Vista (39.9 mg/L)	122 St. & 124 Ave. (65 mg/L)
1976	Groat (42 mg/L)	40 St. & 96 Ave. (28 mg/L)
1977	Dawson (42 mg/L)	170 St. & Mayfield Rd. (155 mg/L)
1978	Groat (6 mg/L)	122 St. & 125 Ave. (19 mg/L)
1979-80	Groat (530 mg/L)	Grierson (107 mg/L)
	50th St & 125 Ave (324 mg/L)	50th St & 10th Ave. (35 mg/L)
1980-81	Groat (<1 mg/L)	50th St & 10th Ave. (<1 mg/L)
	50th St & 125 Ave (<1 mg/L)	
1981-82	Groat (1 mg/L)	50th St & Yellowhead (1 mg/L)
1982-83	from this year on oil and grease measures on the snow dumps were abandoned in favor of river sampling upstream and downstream	

[1] C_1 to C_{30} hydrocarbons = 3 mg/L
[2] C_1 to C_{30} hydrocarbons = 19 mg/L

the river. The data indicate that snow dumps contribute substantial quantities of metals, chlorides, and suspended matter to the North Saskatchewan River. Mass quantities for various metals from the Groat site are summarized in Table 3 for the 1980-81 and 1981-82 winters. This data indicates that snow dumps contribute high quantities of several elements which are aesthetic or general quality factors, including: calcium, magnesium, sodium, potassium, iron and manganese. Of the metals considered for primarily health reasons, chromium and lead values are noteworthy. However, data comparing total metals with soluble metals confirms that virtually all of these metals, except sodium, are present in particulate form (Table 4).

Detection limits are poor for some metals (e.g.lead) but the trend is still valid and is not unexpected. This situation means that metals will tend to deposit in river sediments under low flow conditions and that they will be removed with other turbidity in the normal water treatment process.

The importance of good turbidity removal in this context is noteworthy. Consequently, the metal loadings from the snow dumps are mainly relevant to river ecosystem quality corresponding to the potential buildup of metals in the sediments. Available data indicates that metals are not a primary issue in drinking water quality under the conditions which prevail in Edmonton.

Table 3 Contaminant Mass Values for Groat Snow Dump[39]

Element	1980-81 (kg)	1981-82 (kg)
Arsenic	0.6	<0.2
Cadmium	1.3	1.3
Calcium	29000.	72000.
Chromium	8.5	180.
Cobalt	13.	9.9
Copper	13.	22.
Iron	24000.	10000.
Lead	140.	210.
Magnesium	5500.	12000.
Manganese	1400.	1400.
Mercury	.003	<.009
Nickel	18.	32.
Potassium	170.	3950.
Sodium	16000.	56000.
Zinc	76.	110.

Table 4 **Contaminant Distribution (total vs dissolved) Groat Snow Dump Site**[39]

Element	1980-81 (mg/L)		1981-82 (mg/L)	
	Total	Dissolved	Total	Dissolved
Arsenic	0.041	n/a	<0.005	n/a
Cadmium	0.09	<0.05	0.03	<0.01
Calcium	1990.	71.3	1660.	50.
Chromium	0.58	<0.1	4.13	<0.10
Cobalt	0.89	<0.5	0.23	<0.05
Copper	0.86	<0.5	0.50	0.01
Iron	1620.	<0.5	240.	<0.10
Lead	9.74	<2.	4.74	<0.25
Magnesium	376.	18.5	270.	4.6
Manganese	95.1	<0.5	33.	0.13
Mercury	0.0002	n/a	<0.0002	n/a
Nickel	1.21	<0.5	0.74	<0.10
Potassium	11.9	1.7	91.	10.
Sodium	1100.	420.	1290.	1210.
Zinc	5.18	<0.1	2.53	0.01

2.6.2 Rural Runoff

Runoff from upstream rural areas can pose a concern with regard to agricultural chemicals (pesticides and fertilizers) and natural organic materials, commonly referred to as humic substances, arising from decaying vegetation and muskeg areas.

2.6.2.1 Agricultural Chemicals. Most of the agricultural activity upstream of Edmonton occurs downstream of Drayton Valley, a stretch of approximately 200 km. Upstream of Drayton Valley cleared land becomes much less prevalent. Even in the downstream reach, land within site of the river is not intensively cultivated.

Fertilizers are mainly applied for nitrogen, phosphorus and potassium. Of these, only nitrogen can normally pose a risk to a water supply. Nitrogen in the form of nitrates or nitrites are limited by the Canadian Drinking Water Guidelines[10]. However, data reviewed in section 2.2 provide no evidence of nitrogen levels in the North Saskatchewan River which would be a concern for a drinking water supply. Comparison with other raw water sources in section 2.4 indicated that the North Saskatchewan River was a good water source with respect to nitrogen.

A wide range of pesticides and herbicides are commonly used in agriculture. Because these chemicals are designed to be toxic to animal or plant life, they naturally pose a concern for water supplies. Based upon this concern, monitoring for 24 pesticides and herbicides was initiated for the North Saskatchewan River in 1978. Reynoldson[16] reviewed the data from 1978 to 1981. He found that for the Devon site, only five of these chemicals were ever detected and only two were found routinely, alpha-BHC and 2,4-D. These findings are summarized in Table 5.

Alpha-BHC has no insectidal properties and is found as an impurity in some pesticide formulations according to Gummer.[40] He reported this chemical as occurring widespread throughout the prairies, even in remote watersheds. Although no specific drinking water guideline is available for this compound, the measured concentrations are extremely small.

The herbicide 2,4 - D is the active ingredient in most lawn care products for broadleaf weed (including dandelion) control. Accordingly, this herbicide is widely used. Reynoldson[16] found that concentrations of this chemical did increase substantially downstream of Edmonton. Actual raw water values found at Devon are well below the Canadian Drinking Water Guideline for a treated water supply.

Monitoring for 35 toxic organic chemicals has continued at Devon during the period from 1982 to 1985. These data[14], collected by Alberta Environment and stored on the Naquadat data base, is summarized in Table 6. For this period, only alpha and gamma-BHC and 2,4-D were detected, again at extremely low concentrations. Although the sampling done for pesticides analysis is not very frequent, the available data provide no evidence of an existing threat to the Edmonton water supply from upstream agricultural use of pesticides.

Table 5 Occurrence of Pesticides in the North Saskatchewan River at Devon 1978-81[14]

Pesticide	Raw Water Mean (ug/L)	 Maximum (ug/L)	Canadian Drinking Water Guideline (ug/L)
gamma-BHC	<0.001	0.001	4
alpha-BHC	<0.004	0.007	not specified
2,4 - D	<0.007	0.04	100
2,4,5 - T	<0.003	0.02	10
2,4 - DP	<0.004	0.01	not specified

Table 6 Monitoring for Toxic Organic Chemicals North Saskatchewan River at Devon, 1982-85[14]

Chemical	Number of Samples	Number of Values below Detection	Detection Limit mg/L	Mean Value mg/L	Maximum Value mg/L
Aldrin	12	12	0.001	<0.001	<0.001
Alpha-BHC	12	1	0.001	<0.003	0.005
Alpha-chlordane	12	12	0.003	<0.003	<0.003
Gamma-chlordane	12	12	0.002	<0.002	<0.002
o,p-DDT	12	12	0.001	<0.001	<0.001
p,p-DDD	12	12	0.002	<0.002	<0.002
p,p-DDT	12	12	0.004	<0.004	<0.004
Dieldrin	12	12	0.002	<0.002	<0.002
Alpha-Endo-Sulfan	12	12	0.001	<0.001	<0.001
Beta-Endo-Sulfan	12	12	0.003	<0.003	<0.003
Endrin	12	12	0.002	<0.002	<0.002
Heptachlor	12	12	0.001	<0.001	<0.001
Heptachlor Epoxide	12	12	0.002	<0.002	<0.002
Gamma-BHC (Lindane)	12	9	0.001	0.001*	0.001
p,p-Methoxy-Chlor	12	12	0.010	<0.010	<0.010
2,4-DP	9	9	0.004	<0.004	<0.004
2,4-D	13	11	0.004	0.005*	0.008
2,4,5-T	13	13	0.002	<0.002	<0.002
2,4-DB	9	9	0.009	<0.009	<0.009

Table 6 continued

Chemical	Number of Samples	Number of Values below Detection	Detection Limit mg/L	Mean Value mg/L	Maximum Value mg/L
Mirex Total	12	12	0.001	<0.001	<0.001
Aroclors Total (PCB'S)	13	13	0.002	<0.002	<0.002
Aroclor 1254 (PCB'S)	7	7	0.002	<0.002	<0.002
Aroclor 1260 (PCB'S)	7	7	0.005	<0.005	<0.005
Guthion	4	4	0.50	<0.50	<0.50
Azinphosethyl	4	4	0.20	<0.20	<0.20
Imidan	4	4	0.20	<0.20	<0.20
Crufomate	4	4	0.20	<0.20	<0.20
Parthion	4	4	0.02	<0.02	<0.02
Parathion-methyl	4	4	0.02	<0.02	<0.02
Malathion	4	4	0.05	<0.05	<0.05
Fenchlorphos (Ronnel)	4	4	0.02	<0.02	<0.02
Diazionon	4	4	0.02	<0.02	<0.02
Phorate	4	4	0.02	<0.02	<0.02
Ethion	4	4	0.02	<0.02	<0.02
Carbo-phenothion	4	4	0.02	<0.02	<0.02

Because many of the organic chemicals which present a toxic concern are also poorly water soluble, there is reason to expect that higher concentrations of chemicals may be found in aquatic organisms or on sediment particles. A survey[41] was conducted by the Alberta Environmental Center (AEC) on fish from the North Saskatchewan River downstream of Edmonton. This study found detectable residues for several chlorinated pesticides and PCBs but did not find detectable residues for phenoxy herbicides (e.g. 2,4-D) or organophosphorus pesticides. Since the sampling sites were downstream of Edmonton, it is not possible to conclude anything about upstream conditions. However, the values found were not remarkable compared with values reported for fish at other locations in the world.

Another study AEC study[42], focusing on methoxychlor and 2,2-bis(p-methoxyphenyl)-1,1-dichlorethylene residues in fish, included 515 fish samples from the North Saskatchewan River. Sample sites included Rocky Mountain House, Drayton Valley and Devon. No detectable quantities of either chemical were reported for any of the samples.

A study[43] on PCB partitioning in the North Saskatchewan River included 4 sites upstream of Edmonton. Estimates of waterborne concentrations of PCBs, derived from fish residue values at these sites predicted that waterborne PCBs should be below current detection limits. This prediction is consistent with the monitoring results in Table 6.

A federal study[44] on occurrence and pathways for toxic chemicals in the North Saskatchewan River concluded that the trace organic chemicals commonly pursued in monitoring studies will not usually be found dissolved in water, but rather will be associated with suspended sediments. That finding indicates that good water treatment, producing low final turbidity values, should protect the treated water supply from trace organic chemicals associated with suspended sediments. Once again, the importance of good turbidity removal is noteworthy.

Finally, a discussion about trace chemicals in the North Saskatchewan River cannot be complete without mention of mercury residues in fish. An AEC study[45] found sufficient mercury residues in North Saskatchewan River fish to provide a warning against consumption of such fish by pregnant women. The source of the mercury was not verified. The report did speculate that since the fish were migratory and no sources could be identified in Alberta, despite considerable sampling, the fish likely acquired the mercury from known sources in Saskatchewan.

2.6.2.2 Natural Organic Carbon. Decaying vegetation such as fallen leaves ultimately leads to large molecular weight organic materials. These can be broadly called humic material.[46] The natural decay process ultimately leads to the formation of soil organic matter or soil humus. Freshly decayed vegetation will also contain a complex spectrum of smaller molecular weight organic chemicals.

All natural surface waters contain measurable quantities of organic carbon from these types of natural sources. When concentrations become high enough, the water will display visible color. Thus, water from a swamp or from a muskeg region displays a yellowish brown color which is caused by humic materials.

These natural organic materials can be measured in water by a number of different means. Dissolved organic carbon will measure any soluble organic compounds present in water. In the absence of other significant sources of organic material, dissolved organic carbon (DOC) provides a good measure of waterborne humic materials. These can also be detected in water by colour, which is a measure of the visible light absorbed by the water containing humics.They can also be detected by ultraviolet light absorbance.

Humic materials can cause problems in a raw water source. At sufficient concentrations, humic materials provide a visible colour to the water which consumers are likely to find objectionable. Because these compounds are oxidizable they will react with disinfectant chemicals such as chlorine,

chlorine dioxide and ozone. This reaction causes a demand for the disinfectant. That demand detracts from the disinfectant's primary role in killing micro-organisms unless the dose is raised to exceed the demand. As well, various oxidation products, which are new organic chemicals, can be formed by reactions between the disinfectant chemical and the humic molecules. The earliest serious concern in this regard arose with the findings of Rook[47] that various haloforms, most notably, chloroform, can be produced when water containing humic materials is chlorinated.

For the foregoing reasons, high levels of humic materials are undesirable in a raw water supply and they will cause a variety of treatment and finished water quality problems.

Water contributed to the North Saskatchewan River from glacial melt is very low in dissolved organic carbon and corresponding humic materials. Clean snow melt and fresh rainfall will also be low in dissolved organic carbon. However, water draining from swampy or muskeg regions or water which has been in direct contact with decaying vegetation can exhibit very high levels of organic carbon. The situation for the North Saskatchewan River is clearly depicted in Figures 5.1 to 5.6. These plot the increase in dissolved organic carbon in the North Saskatchewan River from the headwaters downstream to Devon, along with DOC values for some major tributaries, at various sampling dates from February to October 1985.[14]

Fig. 5.1 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River. : Sept. 3 or 4, 1985**

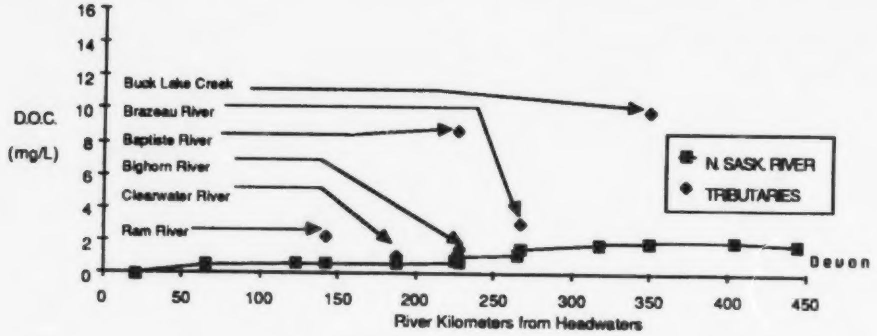


Fig. 5.2 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River for Oct. 2, 1985**

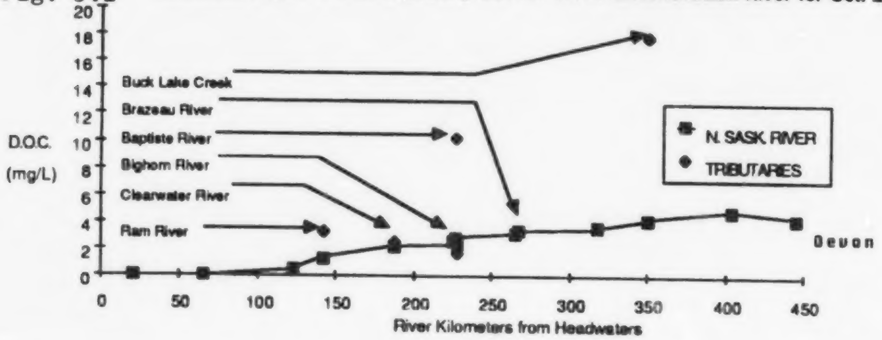


Fig. 5.3 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River : Feb. 4, 1985**

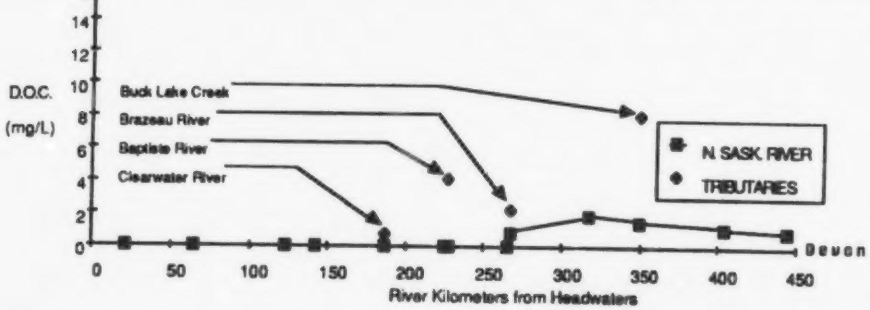


Fig. 5.4 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River : May 6, 1985**

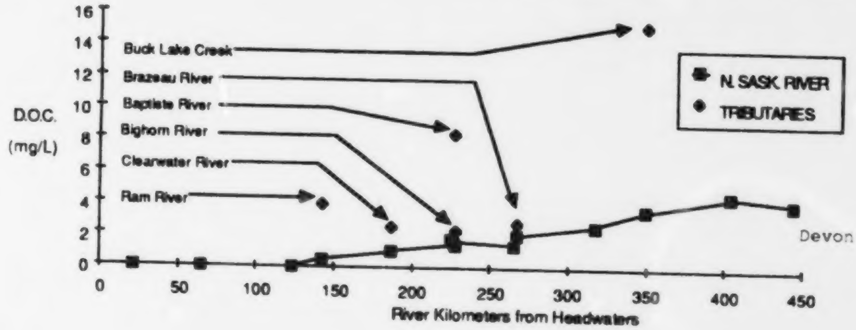


Fig. 5.5 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River. : June 11, 1985**

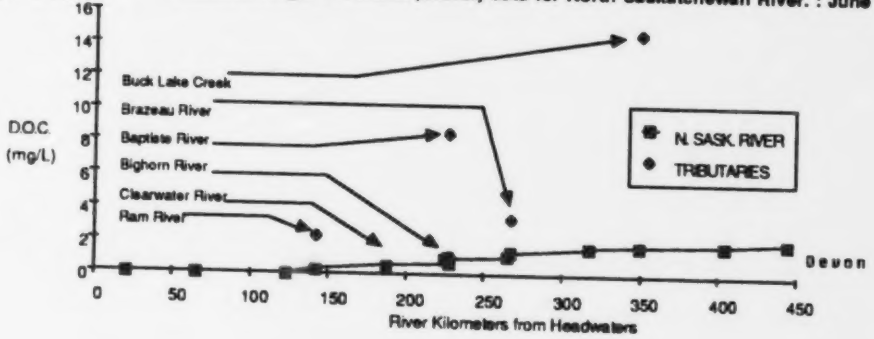
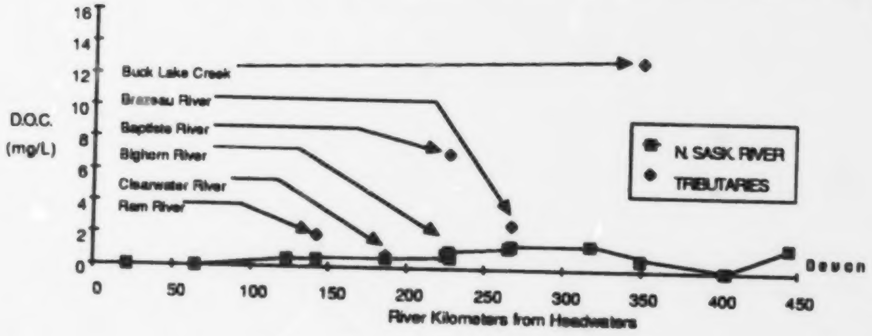


Fig. 5.6 **Dissolved Organic Carbon (D.O.C.) data for North Saskatchewan River : July 9, 1985**



Samples collected on May 12, 1986, during helicopter surveillance of the river up to the Brazeau reservoir indicated DOC values[48] of: 2.8 mg/L for the Brazeau River, 11.3 mg/L for the Nordegg River, 18.0 mg/L for Rose Creek and 37.7 mg/L for Tomahawk Creek.

These contributions of high dissolved organic carbon come from natural runoff sources provide serious degradation of the North Saskatchewan River as a raw water source when they dominate over flow which is low in dissolved organic carbon. The dynamics of upstream thaw which determine the relative contributions from low and high organic sources are likely to vary with varying annual thaw patterns. The routine water quality monitoring data available on the North Saskatchewan River do not currently provide a sufficiently detailed record of this phenomenon to assess annual variablity. However, challenges to Edmonton's water treatment processess can be expected when high dissolved organic carbon levels occur in the North Saskatchewan River.

2.6.3 Monitoring Program - 1986.

The available data on the nature of spring runoff and its effect upon raw water supply at the Rossdale and E.L.Smith water treatment plants did not adequately document the water treatment problems which had been experienced. Consequently, Edmonton Water and Sanitation mounted a major sampling program which was initiated with the rapid thaw on February 25, 1986. Samples were submitted to Enviro-Test Laboratories for chemical analysis and to the Provincial Laboratory of Public Health for microbiological testing. This program involved collection of three 1 hour composite samples a day at the locations listed in Table 7. Sample times were calculated to account for river travel time and flow through the treatment plants. In this way, values measured at various downstream locations could be compared to corresponding upstream locations. Because of the intense nature of the sampling program, parameters were judiciously selected to maximize useful information on the water quality problems caused by runoff. The analytical procedures employed by Enviro-Test Laboratories are summarized in Appendix B.

Because of the concerns with the health implications and the taste and odor of treated water during runoff, attention was focused on organics. Two parameters were measured by gas chromatography, purgeable organics and extractable organics. The former would be indicative of lighter molecules which would readily vaporize from water and might be implicated in the odor of water. The latter would be indicative of larger,

Table 7 1986 Monitoring Program

	Sample Times Each Day		
Sites	1	2	3
E.L. Smith-raw	07:00	11:00	15:00
E.L. Smith-treated	10:30	14:30	18:30
Groat Sewer	13:30	17:30	21:30
U of A - raw	14:00	18:00	22:00
Rossdale-raw (north bank)	14:30	18:30	22:30
Rossdale-raw (30.5m off n.bank)	14:30	18:30	22:30
Rossdale-treated	19:30	23:30	03:30

less volatile organic molecules which could be solvent-extracted from water (at low pH to recover phenolic and acidic compounds).

The humic materials described earlier would not report by either of these methods. However, other smaller, natural organic acids, for example, would report as extractable organic compounds. Volatile hydrocarbons and solvents would report in the purgeable organic analysis. Medium and larger molecular weight hydrocarbons would report in the extractable organics analysis.

In order to account for the organic compounds, including the natural humic materials, which would report as neither purgeable nor extractable organics, dissolved organic carbon was monitored. This parameter would correspond to all of the extractable organic carbon plus the humics and other non-extractable organics.

Chlorides provide an excellent tracer of soluble inorganic substances. They will provide an excellent marker of urban runoff and snow dump meltwater because of the contribution of road salt. Likewise, they will arise from sanitary sewage. Upstream concentrations of chlorides in the North Saskatchewn River are very low. Conductivity provided a convenient check on the accuracy of chloride measurements.

Turbidity provides a rapid measure of the presence of fine suspended matter in water which will arise from both natural and urban runoff. Fluctuations in turbidity will challenge the coagulation-flocculation process to provide optimum clarification prior to water filtration.

Results from the spring 1986 monitoring program are depicted in Figures 6.1 to 6.7. The differences in purgeable organic compounds between the E.L.Smith and Rossdale sites during the sample period are shown in Figure 6.1. Clearly, purgeable organics were not a factor at Smith but they were highly variable at Rossdale and the U of A site which is also impacted by urban sources. Monitoring of discharges from the Groat sewer over the same time period was combined with flow estimation to generate estimates of the mass rate of purgeable hydrocarbon addition from the sewer. This is plotted on Figure 6.2 along with the concentrations observed at the Rossdale intake. The peaks do not coincide as might be expected. However, they appear to be uniformly out of phase.

The offset in peaks is likely due to an under-estimation of travel time from the Groat Sewer to the Rossdale intake. These were selected using average river velocities for anticpated river flows. However, the bank velocity which would apply to the Groat sewer plume would be substantially lower, corresponding to a longer travel time. Notwithstanding the possible mismatch in sample times, Figure 6.2 does indicate that substantial quantities of purgeable hydrocarbons were being discharged from the Groat sewer along the north bank. Their appearance at the Rossdale intake is virtually inevitable.

Fig. 6.1. Purgeable Organics at Intakes (February 25 to March 5, 1986)

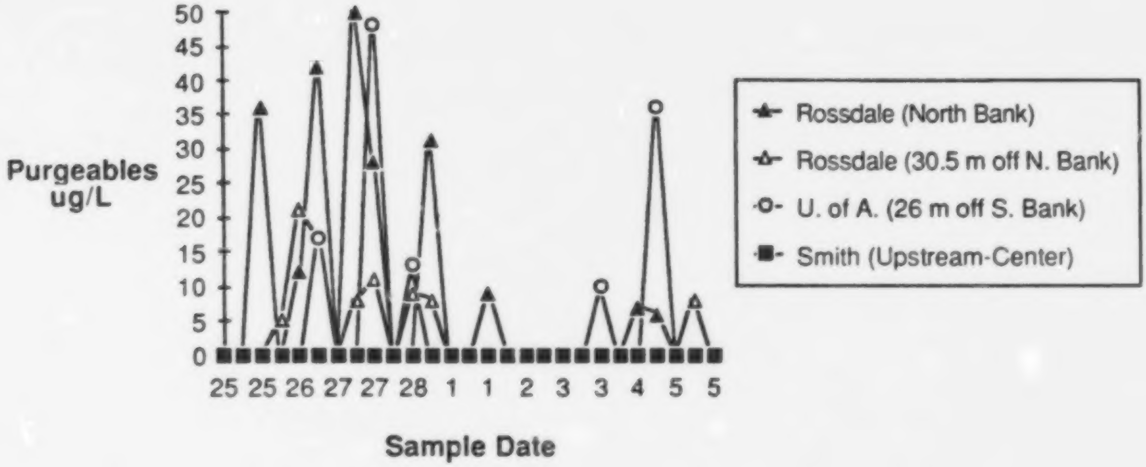
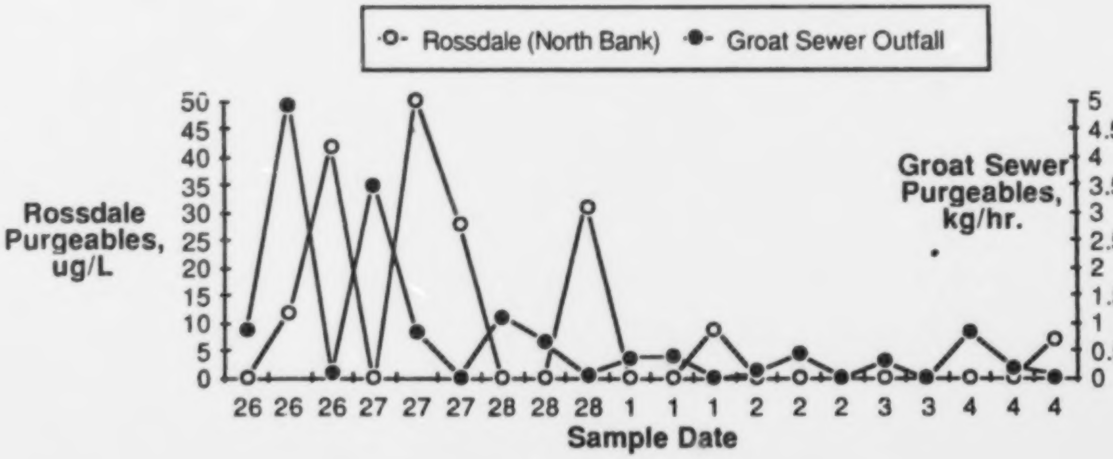


Fig. 6.2. Rossdale Intake and Groat Sewer Discharge - Purgeable Organics (February 26 to March 4, 1986)



The data also indicate that the old Rossdale intake, 30.5m off the north bank, and the University of Alberta intake, 26m off the south bank, are also subjected to urban runoff influences. These other sites are less likely to be influenced by the Groat sewer so their contamination would likely have arisen from some of the many other outfalls.

Figure 6.3 shows the differences in extractable organics between the Smith and Rossdale intakes. The obvious influence of urban contamination between Smith and the city center (Rossdale and U.of A.) sites is even more apparent for extractable organics. Figure 6.4 shows the mass discharge comparison for the Groat sewer and the same time offset occurs.

Figure 6.5 shows the differences in dissolved organic carbon between the various water intakes. In this case, a somewhat different pattern emerges. Until March 1, the influence of urban sources on the city center intakes is apparent. However, on March 1, dissolved organic carbon rises sharply at Smith and is followed closely by the city center plants. Because humic materials would report as dissolved organic carbon, the rise at all intakes was likely the result of upstream rural runoff. This theory is supported by the monitoring conducted by water treatment plant staff for UV absorbance, a good indicator of humic materials. Ultraviolet absorbance (in units calibrated against a laboratory humic acid standard) is plotted in Figure 6.6. This clearly documents a sudden rise on March 1.

Fig. 6.3. **Extractable Organics at Intakes (February 25 to March 4, 1986)**

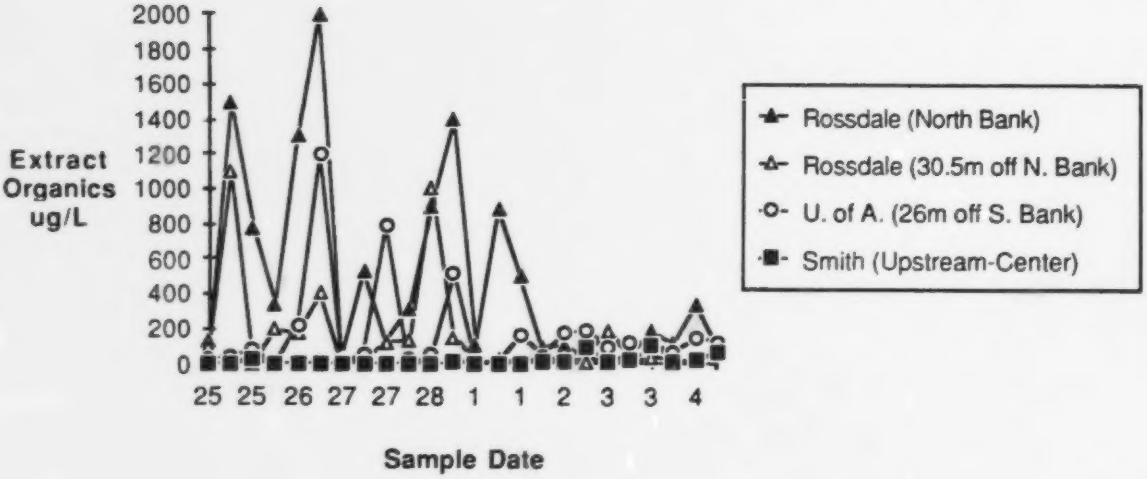


Fig. 6.4. **Rossdale Intake and Groat Sewer Discharge - Extractable Organics (February 26 to March 4, 1986)**

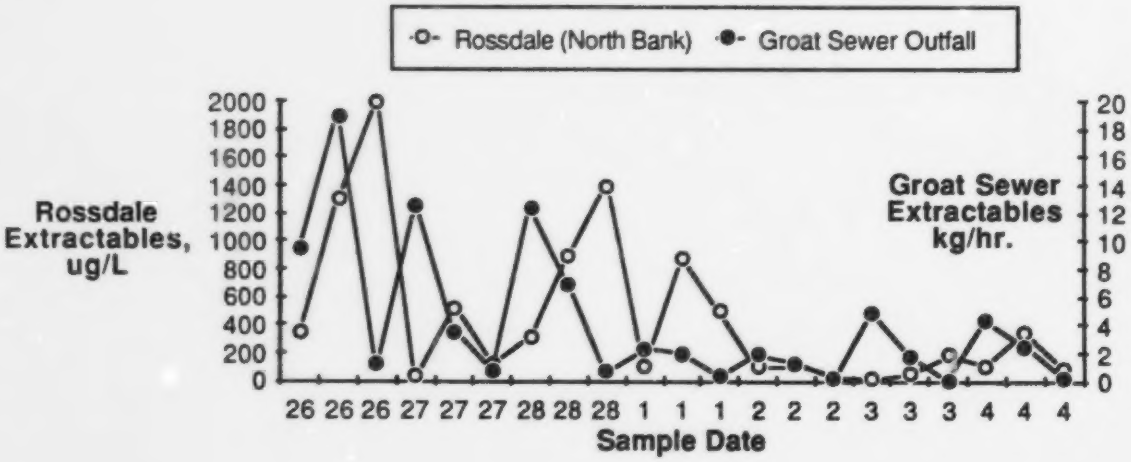


Fig. 6.5. **Dissolved Organic Carbon at Intakes (February 25 to March 5, 1986)**

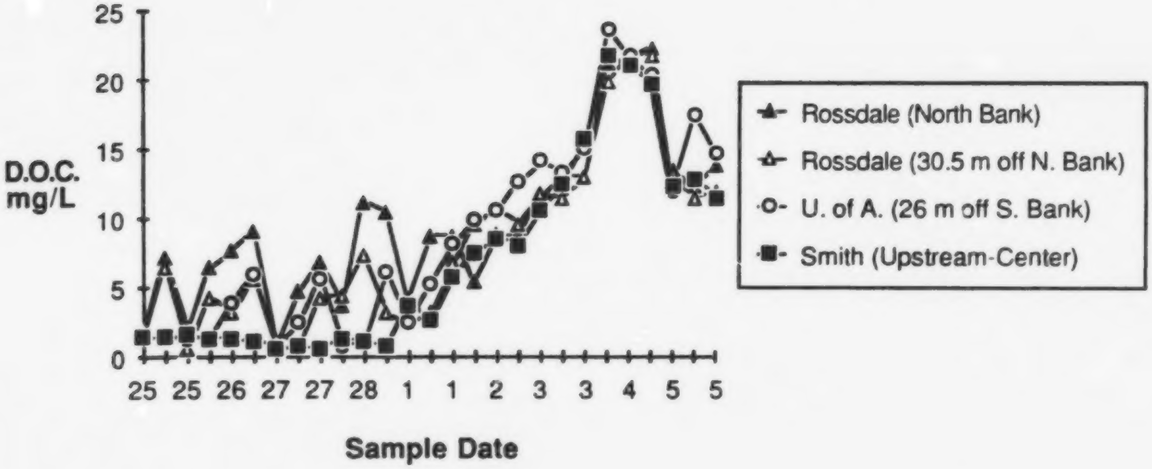
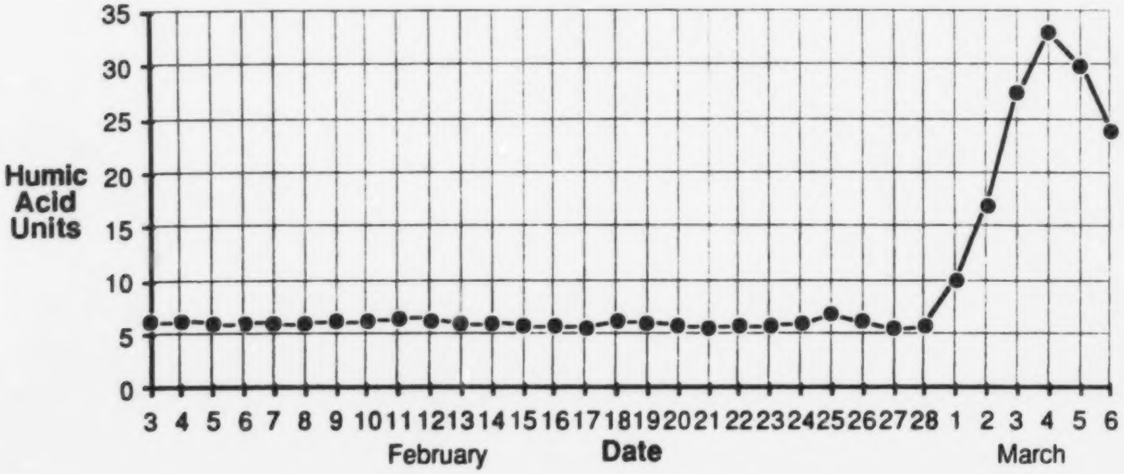


Fig. 6.6. **Relative concentration of Humic Acids in Raw Water at the Rossdale W.T.P. (Feb. 3 to March 6)**



Chlorides (Figure 6.7) provide another element to indicate that urban runoff had been largely exhausted during the exceptionally warm weather from February 25 to March 1. The chlorides indicate only a minor influence on the city center intakes during the second week. For comparison with the organic parameters, the chlorides mass discharge from Groat storm sewer is plotted with the Rossdale intake (north bank) in Figure 6.8. This also shows the out of phase character seen with the organic parameters, but it appears that Rossdale may be influenced by other sources to a greater degree than was apparent for the organics.

Finally, turbidity (Figure 6.9) indicates the complicated treatment challenge experienced by Rossdale, compared with the Smith intake. These rapidly varying turbidity values would make optimization of the coagulation-flocculation process much more difficult at Rossdale.

All of the solvent extracts from the extractable and purgeable organics samples were retained for later review and selected analysis by gas chromatography - mass spectrometry (GC/MS). The data from the GC/MS runs selected is summarized in Tables 8 and 9.

Table 8 compares GC/MS analysis of a Groat Road storm sewer sample and a Rossdale (north bank) intake sample. These samples were selected for GC/MS analysis because they showed the high overall responses on the GC runs for extractable organics determination, but the qualitative pattern was similar for samples at both sites during the early stages of

Fig. 6.7. **Chloride at Intakes (February 25 to March 5, 1986)**

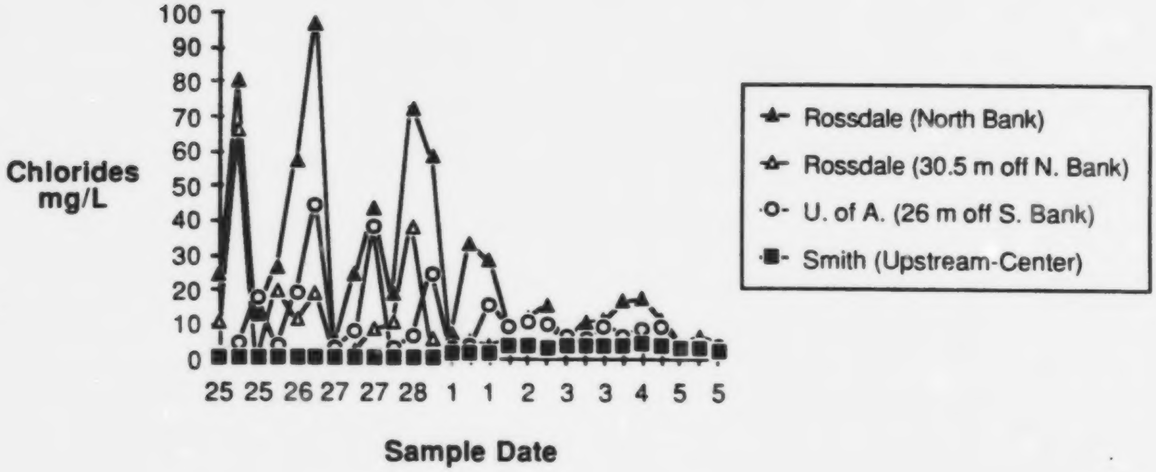


Fig. 6.8. **Rossdale Intake and Groat Sewer Discharge - Chloride (February 26 to March 4, 1986)**

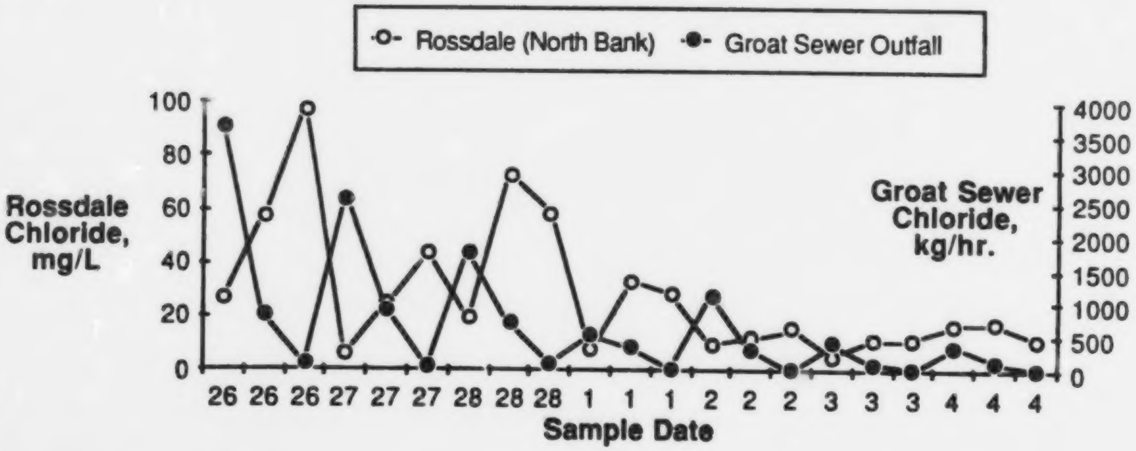


Fig. 6.9. **Turbidity at Intakes (February 25 to March 5, 1986)**

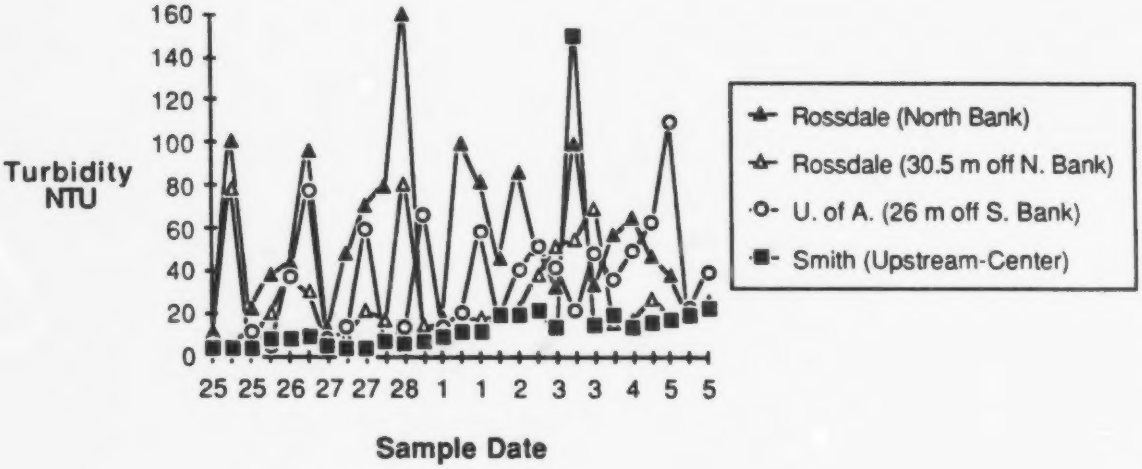


Table 8 Compound Classes Identified

Compound Class	Groat Storm Sewer February 25, 1986 ug/L (ppb)	Rossdale - Raw (north bank intake) February 25, 1986 ug/L (ppb)
paraffins and olefins (hydrocarbons)	13910	1690
aromatic hydrocarbons	640	65
long chain acids	-	240
others	450	4
total extractables	15000	2000

Table 9 Compounds Identified with Reasonable Confidence

Approximate Concentrations, ug/L(ppb), March 3,1986

Compound	E.L.Smith raw	E.L.Smith treated	Rossdale raw north bank	Rossdale treated
butanoic acid	17	9	0.5	-
2,3 dichloro-2methylbutane	-	-	-	1
cyclohexane-carboxylic acid	2	4	12	1
benzoic acid	37	31	-	20
benzeneacetic acid (phenylacetic)	17	12	3	5
benzenepropionic acid	11	8	2	2
methyl phenol isomers	13	-	1.2	-
ethyl phenol isomers	4	-	1.4	-
benzeneacetaldehyde	6	4	-	1
benzeneacetonitrile	-	1	-	1
tetrahydrotrimethyl-benzofuranone	-	1	-	1
nanonoic acid	-	-	-	1
total extractables	110	82	20	33

the urban runoff (February 25 to 27). These analyses confirm that substantial quantities of hydrocarbons were being discharged from the storm sewer and similar types of hydrocarbons were being seen at the Rossdale (north bank) intake. That finding only confirms what would be expected, but earlier studies on the impacts of storm runoff on the Rossdale intake had not demonstrated the similarity of storm sewer source and raw water at the intake.

The corresponding gas chromatograms for these two samples are provided in Figures 7.1 and 7.2. Because these traces are automatically sized to keep the largest peak at full scale, a decrease in the prevalence of the sharp peaks between about scan #17 and #23 on the Groat sample leads to the apparent enhanced size of the hump on the Rossdale intake sample. The peaks which have been reduced in the Rossdale sample would be longer chain normal alkanes which become increasingly water insoluble with increasing chain length.

The normal alkanes can be recognized by the even spacing between large peaks. Because the Rossdale intake is submerged and because insoluble alkanes would tend to agglomerate into droplets which would tend to float as a slick, a lower intake concentration of the alkanes, relative to more soluble species such as the olefins and aromatics, would be expected. Unfortunately, the alkanes which may bypass the intake are the least odorous of the hydrocarbons. Under the circumstances, it is clear that the Groat Road storm sewer was contributing hydrocarbons to the Rossdale water intake.

Fig. 7.1. Gas chromatograms of water samples taken from the Groat storm sewer discharge.

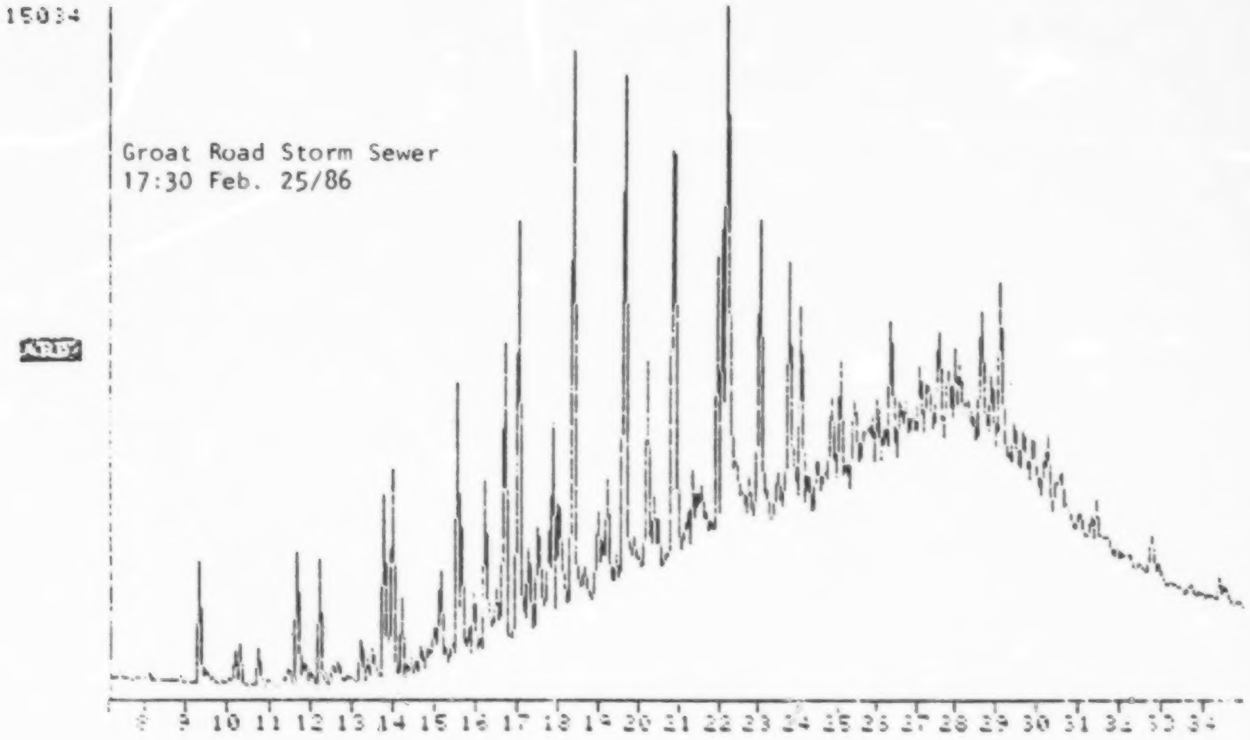


Fig. 7.2. Gas chromatograms of water samples taken from the Rossdale water treatment plant raw water intake (north bank).

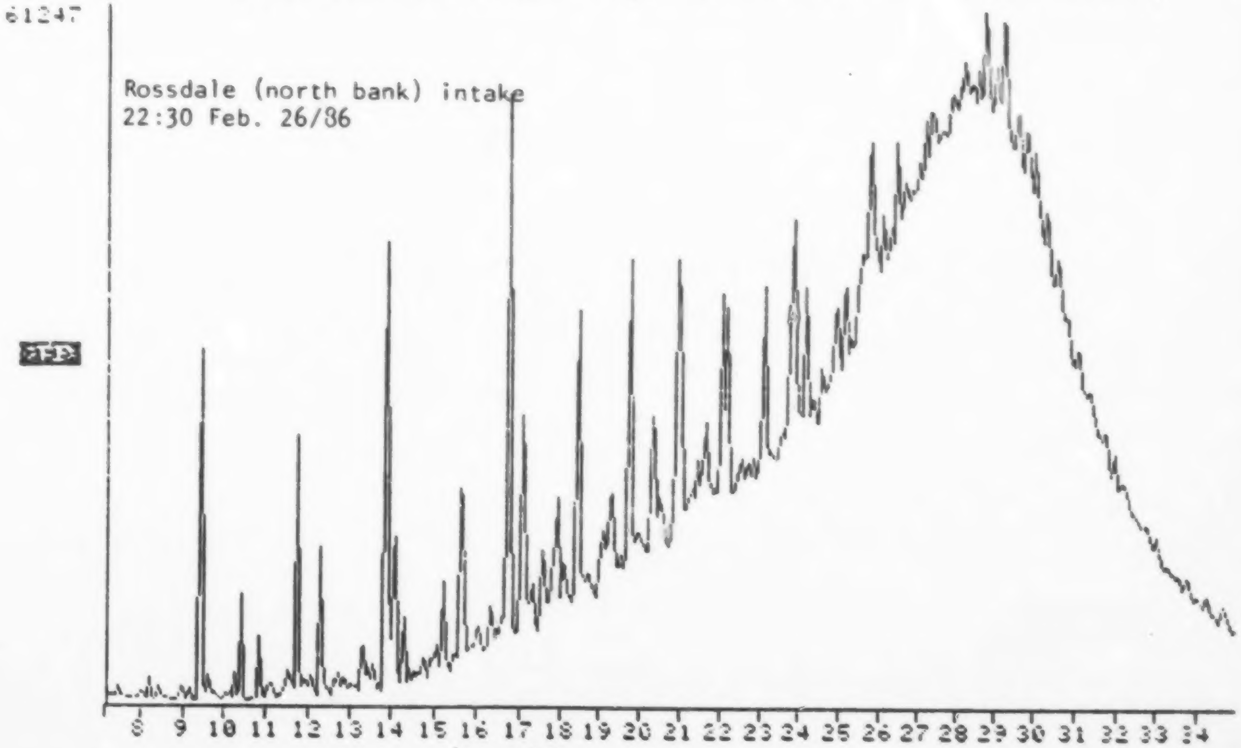


Table 8 also indicates the presence of organic acids at the Rossdale intake which are not noted for the Groat storm sewer. These are more likely a reflection of vegetation and hydrocarbon biodegradation products. They may have also have been present in the Groat sample but could have been masked somewhat by the very strong hydrocarbon response.

After March 1, upstream runoff was seen to dominate over the urban runoff seen the previous week. This difference is reflected in the nature of organic compounds identified by GC/MS at the Rossdale and Smith plants (Table 9). The compounds are generally characteristic of degradation products from vegetative matter. These are distinct from the humic materials which appear coincidently, in that the degradation products observed are smaller molecular weight compounds which are available for further biological processing. The humic materials, which represent an endpoint of the overall degradation process, are too large and too poorly extracted to report in the gas chromatographic analysis for extractable organics.

A better understanding of the nature of the organic carbon carried by upstream spring runoff would be useful for developing treatments to achieve carbon removal. A preliminary attempt at characterizing this material was undertaken by the Alberta Research Council for this study. They concentrated 100 L of river water through ultrafiltration membranes and freeze-dried the organic matter obtained. This material was subjected to ultraviolet, infrared and nuclear magnetic resonance spectroscopic analysis. Results, summarized in Appendix C, indicate that the organic material is characteristic of waterborne humic substances.

The significance of the organics identified is a major issue itself. Some factors are covered with the discussions in section 3.5 and 3.6 but health issues are covered by the reports by the Canadian Center for Toxicology and by Dr. Bull. At this point it is sufficient to clearly recognize that the spring 1986 monitoring program documented the occurrence of two types of runoff. The urban runoff affects the Rossdale plant exclusively while the rural runoff affects both Smith and Rossdale almost equally.

2.7 Spills

Water intakes on a river source are vulnerable to upstream spills of materials which may adversely affect water quality. Such occurrences are far from hypothetical. A summary[49] of spill occurrences prepared by the laboratory staff at the Edmonton water treatment plants listed a total of 31 events over the period from April, 1981 to November, 1984. This record increased from only 1 case for 1981 to 14 cases for 1984. Such a dramatic increase seems to be more likely caused by better record-keeping than an actual increase in number of events. That supposition is supported by the observation that such statistics appeared for the first time in the 1983 Water Treatment Plant Annual Report.[37] Regardless of any uncertainty about the adequacy of event reporting prior to 1983, there is clearly evidence from recent years that spill events are common.

The Edmonton Water Treatment Plants have responded to the onslaught of spills by instituting a volatile organics analysis by purge and trap gas chromatography. The procedure has evolved from a very rudimentary practice to the point where it could now provide useful quantitative records of the volatile organics experienced in water at the plant. The procedure has been used to warn of spill conditions and experience has shown a reasonable correlation between the occurrence of unknown peaks on the chromatograms and the occurrence of complaints about taste and odor. Consequently, the plant has developed an action plan which calls for progressive addition of powdered activated carbon in response to increasing levels of volatile organics. In the extreme, the plan can lead to a plant shutdown in response to very bad water conditions. Overall, this approach has been a positive approach to the poor water conditions frequently experienced at Rossdale.

Consideration of spill potential will be divided into two categories, upstream and urban, for the purpose of discussion.

2.7.1 Upstream Spills

Any human activity in the river basin upstream of Edmonton could provide some potential for spills which could reach the North Saskatchewan River. Depending upon the location, nature and magnitude of the spill, the Edmonton water intakes could be adversely affected. The existing population in the upstream drainage basin is small. That factor alone substantially reduces the probability of spills from domestic or municipal sources. Only six highway river crossings are mapped between Edmonton and Rocky Mountain House, approximately 300 km upstream from Edmonton. Of these, 4 are primary highway crossings (Devon, Drayton Valley and 2 at Rocky Mountain House). This level of exposure to highway traffic is small.

Industrial activity upstream of Edmonton, as noted in section 2.5, is relatively limited. Several small oil batteries were sighted during a helicopter trip up to the

Brazeau Dam. Those located adjacent to the river certainly have potential for hydrocarbons spills which could affect Edmonton's water intakes if allowed to go unnoticed. To that extent, the existence of upstream population centers is advantageous by providing a possibility of spill sighting by upstream observers. There are also several pipeline crossings located upstream of Edmonton with most being clustered in the Edmonton area, near Devon and near Drayton Valley. These could lead to hydrocarbons spills.

Gravel mining and farming operations near the river also provide a potential for spills. Illicit dumping by any party is also possible. An empty chemical drum was observed along the river bank around Genessee Bridge during this year's helicopter surveillance.

The Alberta Department of Health documented a 1960 spill of crude oil into the North Saskatchewan River by a pipeline at Devon. The City of Edmonton listed upstream spill occurrences between 1981 and 1984 to include: an unknown chemical spill at Devon (March 2, 1982) and a gasoline spill at Rocky Mountain House (January 19, 1984).

Considering all the factors which might contribute to upstream spills, including the widespread use of gasoline and fuel in most human activities, hydrocarbon spills seem the most probable. Bulk chemical shipments within this region should be relatively minor. However, Edmonton Water and Sanitation and/or Alberta Environment should perform an inventory of known bulk chemical use activities associated with documented upstream activities. This should include a survey of the types and quantities of pesticides and herbicides marketed to agricultural operations within the drainage basin upstream of Edmonton. The results of such a survey would assist monitoring and contingency planning for upstream chemical spills.

A measure which should be taken to avoid treatment problems arising from upstream spills would be forming a cooperative group with all organizations who withdraw water upstream (Devon, Transalta Utilities, etc.) to guarantee rapid and direct sharing of information concerning unusual water conditions. The potential for continuous monitoring of water quality will be dealt with in a separate report on that issue.

2.7.2 Urban Spills.

As noted in section 2.6, the Rossdale water intake is downstream from 85 storm or combined sewer outfalls which drain most of western and southern Edmonton. This situation provides enormous potential for a wide range of spills to reach the Rossdale intake with little or no advance warning. Of the 31 spill events mentioned in section 2.7.1, 13 were directly associated with storm sewer flows to the river upstream of the intake. The Rossdale plant has no capability to drain poor quality water from the plant, once in the treatment process stream, without shutting down the plant.

Therefore, introduction into the plant of water contaminated by a spill presents major problems.

Impacts on treated water quality at Rossdale have occurred because of a chemical spill to the Groat sewer on at least one occasion. This incident occurred in October, 1979 and is documented in a report[50] by A.Masuda of Alberta Environment. Edmonton drinking water was tainted with a chemical taste during the week of October 1, 1979. A citizen spotted "pink foam" at the Groat sewer outfall on the evening of October 3, 1979. Edmonton Water and Sanitation was contacted to trace the source of the foam because the city is responsible for storm sewer discharges to the river. It was not until October 5 that Alberta Environment became aware that the water treatment plants had been receiving taste and odor complaints and storm sewer samples were taken for GC/MS analysis. The sewer sample indicated a complex mixture of hydrocarbons (alkylated benzenes and naphthalenes) typical of diesel fuel but it also contained 30 mg/L of phenol. The chemical taste experienced with the water supply was attributed to this chemical/ hydrocarbon spill which was transported to the river by the Groat storm sewer. The investigation was unsuccessful in confirming the origin of the spill material.

The foregoing incident and the existing circumstances of the Rossdale intake indicate that it is highly vulnerable to chemical spills which could arise anywhere in the region drained by the storm sewer network. Because of the poor lateral mixing which occurs in the river (see Smith report in this Volume), the Rossdale intake is mainly at risk from left bank (north side at Rossdale) discharges and it is extremely vulnerable to discharges from the Groat Road storm sewer. This network drains residential, industrial and commercial areas and transportation routes. The Groat Road North, Mckinnon Ravine, Quesnell Bridge North and Terwillegar Park sewer systems include proportions of industrial drainage of: 25, 15, 24 and 18% respectively.[32]

Experience has shown that traffic accidents can and do cause spills to the storm sewer system which are seen at the Rossdale intake. Firefighting could also give rise to contaminated discharges. Recent experience in the maritimes with extensive contamination caused by water draining from a pesticide chemical warehouse fire should highlight this possiblity.

Major accidents should usually, with education and cooperation from the emergency response teams, result in warnings being issued to the water treatment plants. The possibility of accidental or intentional waste dumping to the storm sewer system, as occurred in October 1979, presents a much greater risk of tainted water being drawn into the Rossdale water intake.

Given the high degree of vulnerability of the Rossdale intake to chemical spills arising from the storm sewer network, intake protection with a margin of safety appropriate to a public water supply can only be achieved by relocating the Rossdale intake to an upstream location which is not at risk from spills to the storm sewer system. Some protection could be afforded by the use of continuous monitors. This subject is reviewed in a separate report (Hrudey-2, this Volume), but the findings indicate that adequate protection cannot be assured by such measures at this time.

3. TREATED WATER SUPPLY

3.1 System Description

Four individual water treatment plants are located at Edmonton's two water treatment facilities, Rossdale and E.L. Smith (Figure 1.2). Plant #1 was commissioned at Rossdale in 1947, followed by Plant #2 in 1956. These are essentially equivalent plants in design and layout. Plant #3 was added in 1967. Finally, the E.L. Smith plant, designated plant #4, was opened in 1976.

Through staged upgrading at the older plants they are all now using the following processes: alum coagulation, clarification, chlorine dioxide disinfection, fluoridation, rapid sand filtration and chloramine disinfection. Partial softening with lime and recarbonation is practiced at plants 1,2 and 4. Finally, powdered activated carbon can be dosed intermittently at the point of alum addition, as needed, for organics removal. These processes would qualify as complete treatment according to the Canadian Drinking Water Guidelines.[10] Auxiliary treatment, in the form of pre-chlorination, pre-sedimentation and post-chlorination are not practiced in Edmonton. However, the powdered activated carbon and possibly the chorine dioxide could be interpreted as "Other advanced forms of treatment approved by the control agency may be considered equivalent to any of the traditional methods named above.", in accordance with the Canadian

Drinking Water Guidelines.[10] Further details of the Edmonton water treatment plants are discussed in the expert reports by Foley, Huck, Logsdon, Snoeyink and Trussell (this Volume).

3.2 Relation to License Requirements

The Rossdale and E.L. Smith plants are licensed separately under the Alberta Clean Water Act. Their licenses are provided in Appendix D. An assessment of the adequacy of these licenses and comparison with other jurisdictions is provided in the report by Foley (this Volume).

This report must consider whether the Rossdale and E.L. Smith plants have been meeting their license requirements as they relate to treated water quality. For this purpose, the City of Edmonton Water Treatment Plants monthly reports for 1985[51-62] and January to April 1986[63-67], the Annual Reports for 1980 to 1984[68-70,37,30], the Alberta Environment treated water surveys from 1983 to March 1986[71-73] and any relevant special reports available are reviewed.

The main quality requirement specified in the licenses, is the requirement to produce water which meets the Guidelines for Canadian Drinking Water Quality, 1978.[10] In addition, minimum combined chlorine residuals in City reservoirs and in the City distribution system of 1.0 and 0.5 mg/L respectively are required. Finally, fluoride is specified at a monthly average of 1.0 mg/L with an allowable variation of plus or minus 0.1 mg/L and a daily allowable variation of plus or minus 0.2 mg/L.

3.2.1 Review of Monthly and Annual Reports

Before embarking on a critical review of events which occurred some time ago, it is worth recalling that hind sight is 20/20 vision. Current knowledge will necessitate commentary on matters which may have been far from obvious at the time of events. Future reviewers of this document will certainly possess better insight into the issues currently under discussion than may now be possible.

This review should be directed at what can be learned for current and future practices rather than dwelling on past inadequacies. The focus on older events will be mainly on those issues which may provide a lesson (positive or negative), have a current message or a future application.

A general observation is also warranted. The annual reports produced by the Edmonton Water Treatment Plants are much more detailed than required by their Clean Water Act License. Edmonton produces reports for public distribution with considerably more information about their operations than is currently provided by most other cities, including Calgary. Based upon attempts to obtain comparative data for other cities, Edmonton's reporting is remarkably detailed.

1980. The Annual Report[68] reviewed a year of transition. The Guidelines for Canadian Drinking Water Quality, 1978[10] were made available and adopted in mid year. This led to some changes in monitoring requirements. The number of coliform samples recommended by the guidelines[10] dropped from 260 to

150 samples per month (based upon a population of 600,000 served) after 6 months of the year. However, only 51.4% of the specified number of samples were taken by the Local Board of Health and analyzed by the Provincial Laboratory of Public Health.

The color guideline of 15 true color units (tcu), which is set on aesthetic grounds, was exceeded on two days in the spring during highly colored raw water conditions arising from upstream runoff. Values of 38 (also given as 37 elsewhere in the report) and 17 tcu were reported. The turbidity guideline of 5 nephelometric turbidity units (NTU) was also exceeded on two days reaching 8 NTU at Smith and 6 NTU at plants 1&2 at Rossdale. There is a discrepancy between the text of the report and the tables of performance with the latter showing a maximum value of 1.9 NTU for plants 1&2. In explaining the turbidity variances, the report notes that turbidity values can only be measured on grab samples. This assumes that one does not choose to use on-line turbidity monitors which were certainly available and well known in 1980. The report goes on to note that this grab sample could pick up a high value of turbidity because of filter backwashing operations and thereby overestimate the daily average turbidity. This logic fails to recognize the importance of "real time" turbidity values which represent the solids content of the water at any time. Because a high turbidity value could be associated with periodic breakthrough of Giardia cysts or viruses, short term high values are not irrelevant. It would not be necessary to fully contaminate an entire day's production of water to deliver a batch of water containing pathogens. When dealing with pathogens exhibiting a low infective dose (Giardia or viruses) such a situation could presumably lead to sporadic disease occurrence. Looking at turbidity values in this way, the single daily grab sample would be more likely to underestimate the critical peak values of turbidity which could correspond to pathogen breakthrough.

Another aspect concerning turbidity was the institution of turbidity monitoring during the coldest winter months. This was an important step forward because experience has shown that cold, low turbidity waters can pose a substantial risk for Giardia transmission (see Logsdon report, this Volume).

Violations were also experienced with fluoride. The chart depicting fluoride levels showed mainly variance on the low side, reaching as low as 0.36 mg/L versus the target of 1.0 mg/L. Variance on the high side reached 1.14 mg/L. Tabulated values showed two lines of fluoride data. The 1.14 value corresponded to a designation that analyses were performed by Alberta Environment. The other fluoride values showed a maximum of 1.48 mg/L without comment. This value comes very close to the guideline maximum acceptable concentration of 1.5 mg/L , above which dental fluorosis (mottling of teeth) may be expected in some members of the population after sustained exposure. The discrepancy in reporting these higher fluoride values without comment is disturbing.

Overall, the violations for 1980 seem minor considering the heavy runoff apparently experienced that year and limitations imposed by the aging condition of the Rossdale plant. This view is tempered by the view that turbidity compliance can no longer be viewed as primarily an aesthetic aspect.

The number of discrepancies encountered in the brief review of this report suggests an inadequate level of quality control in the report production. Certainly, typographical errors can and will occur, as I am sure will be verified at various points in this report. However, errors such as those mentioned above, in the final tabulation of a year's performance are not acceptable. Such large and easily recognized errors in the Annual Report do not instill confidence in the process of data handling and record keeping upon which the final values must be based.

1981. This was not a vintage year for the Edmonton Water Treatment plants, at least as reflected by the 1981 Annual Report.[52] Reading this report provided an impression of being the first person to have ever read the report in earnest.

The report covers 635 violations of the license conditions. Over half of these (329) are for temperature and are not important for this review. Another 108 were for fluoride, mainly low values. However, the high values were listed as reaching 20.5 mg/L at Plant #3. If that value is accurate, which seems outrageous, it would indicate dangerously inadequate process control. There was no indication of how many other values may have been this high or low or for how long.

Another 94 violations were for pH values above 8.5 which is undesirable but not a serious problem if brought under control. A total of 57 violations of the phenol limit of 2 ppb were also listed. In explaining these in Table 6.15 the report[69] states: "All violations but one (plant No. 4, 9ppb, Sept. 4) less than or equal to 2 ppb over the limit." The limit is 2ppb or 0.002 mg/L. In the summary data Table 6.1, maximum phenol values are quoted as 4.7 mg/L (ppm) for plants 1&2, 5.1 mg/L (ppm) for plant 3 and 8.9 mg/L (ppm) for plant 4. These extraordinary values cannot be reconciled with the previous statement even if one slips the decimal place by a factor of 1000.

Minor color violations occurred on 4 occasions. Turbidity violations numbered 30 in all with maximum values of 9.8 at plants 1&2 and 12 at plant 3. Turbidity values of 2.4 to 10 were reported at Ormsby reservoir from May through October. These values are much too high for the reasons noted in the discussion of the 1980 report (see also Logsdon report, this Volume).

A total of 9 metals violations are blamed on inadequate treatment capability at the existing Rossdale plant. No explanation is offered as to how refurbishment is expected to improve the situation. Several inconsistencies occur. For cadmium, the maximum values are reported as <0.02 mg/L and the minimum as <0.00002 mg/L. The maximum acceptable concentration for cadmium, according to the license is 0.005 mg/L. Although the report explains elsewhere that detection limits sometimes varied, I cannot accept that they would vary by a factor of 1000 in the same lab. Because there is no designation to indicate analyses by an outside lab, I must assume all samples were analyzed in the same lab. Even if such variation could be explained, there seems to be little to commend analyzing for a metal when restricted to a detection limit which is 25 times the maximum acceptable concentration. A similar discrepancy, although not as dramatic, occurred with lead.

Mercury is reported at maximum values of 0.0074 mg/L for plants 1&2, 0.13 mg/L for plant 3 and 0.003 mg/L for plant 4. These compare with the maximum acceptable value specified by the license of 0.003 mg/L. In explaining these high values in Table 6.15 the report[69] observes: "Source of river contamination unknown". A review of Table 6.1 shows maximum river values of 0.003 mg/L at Rossdale and 0.002 mg/L at Smith. The Rossdale maximum for mercury in the raw water certainly cannot explain the high value in the treated water on the basis of "river contamination".

Selenium is reported with maximum values of 2.5 mg/L at plants 1&2, 1.1 mg/L at plant 3 and 0.4 mg/L at plant 4. These values range from 40 to 250 times the maximum acceptable limit in the license. These values are explained in Table 6.15 as: "On one day grab samples revealed levels of 2.5mg/L, 1.1 mg/L and 0.4 mg/L respectively for Plants 1&2, 3,4. Source of contamination unknown."

Immediate resampling is called for when maximum acceptable concentrations are exceeded.[10] Contamination as gross as suggested by these numbers would demand a substantial investigation to re-establish any confidence in sampling and analytical procedures. Similar gross numbers were reported for antimony without comment.

A single high value of 1.7 mg/L was reported for nitrite at plant 3. This occurrence is acknowledged without comment in Table 6.15. The maximum acceptable concentration according to the license is 1 mg/L. A single high value for nitrite is very unusual and the acute nature of health concerns for this parameter certainly warrants some commentary.

About 100 complaints were received in each of February and March compared with other monthly values from 3 to 19. The report notes that there were 10 days of offensive odor at Rossdale plants 1&2 and 3 days for plants 3 and 4.

The license requirement of 1.0 mg/L total chlorine was not met for required chlorine residuals in the Castledowns (0.61 mg/L) and Clareview (0.23 mg/L) reservoirs. That could certainly lead to deterioration in the aesthetic quality of water because of bacterial regrowth and it raises concerns about microbiological safety. On the positive side, the Local Board of Health collected 8% more samples from the distribution system than recommended by the guidelines for bacteriology analysis by the Provincial Laboratory of Public Health.

Overall, the 1981 report[52] reflects an operation which is not adequately under control. The average treated water quality conditions were adequate. Poor conditions, at least if the data is accepted at face value, were unacceptable for a water utility of this size. Treatment and reporting performance at the level reflected by this report would not earn public confidence in the management of their water supply.

1982. Perfomance, at least as reflected in the presentation of the Annual Report[70], improved noticeably in 1982. There were still 643 cases of exceeding license limits, but 336 were for temperature, 68 for pH above 8.5 and 199 for fluorides. Of the latter, 70% were caused by unavailability of the fluoridation chemical, hydrofluosilicic acid. The other 30% were attributed to plant "obsolescence", the same reason given in the 1981 report. No recommendations for dealing with the problems caused by the noted obsolescence were noted in the report.

A detailed record of the dates and values of the fluoride non-compliance was provided (Table 6.19) which was an improvement over 1981. High values of 1.54 mg/L and 2.03 mg/L were reported on July 7 and 8 and 1.50 mg/L on July 24. This continuing poor control of fluoride addition at plant 3 in the year following the extraordinary 20.5 mg/L value (even if we were to assume this was a typographical error, and that it should have been 2.05 mg/L), is not acceptable. Very tight control over fluoride addition is absolutely essential as indicated by the tight limits specified for fluoride in the license.

The phenol limit of 0.002 mg/L was exceeded on 17 occasions. As with fluoride, a more detailed summary was provided in Table 6.20. This indicates that excessive values ranged from 0.003 up to 0.011 mg/L. There were 10 violations at Rossdale and 7 at Smith, where the highest value was recorded.

The turbidity limit was exceeded on three occasions with values of 8.2, 6.1 and 5.8 NTU. These were described as: "short-lived, isolated spikes...Not observed in distribution system (obsolescence of plant)". That logic overlooks the Giardia risk, as mentioned previously. Similar logic was used to explain two color violations. In those cases, the logic concerning averaging is more acceptable.

The manganese limit was exceeded on 3 occasions, reaching a maximum of 0.123 mg/L. This parameter is controlled for aesthetic concerns with staining of plumbing fixtures and laundry. No complaints were received.

The mercury limit was exceeded on two occasions at Rossdale (0.0018 and 0.123 mg/L) and once at Smith (0.0015 mg/L). The only comment offered was "Source unknown". Single high values, particularly when they are 123 times the license limit, suggest the need for immediate followup action on sampling and analytical quality control. No such action was described.

Antimony has been monitored by the City because an objective level of 0.0002 mg/L was set in the Canadian Drinking Water Guidelines.[10] That document declined to set a maximum acceptable concentration for antimony because there was inadequate health data to establish such a standard (see section 2.2.2.1). The suggested objective level (<0.0002 mg/L) is extremely low as was the case for all objective values proposed.[10] The Guidelines state: "This level is interpreted as the ultimate quality goal." They do not represent a level above which health concerns would necessarily arise.

Edmonton reported 25 variances with the objective level with the highest being 0.006 mg/L. These do not constitute violations of the license limit, in my view. The implications of values in excess of the objectives are minor compared with violations of the maximum acceptable concentration. The only significant concern with regard to antimony levels from this report is the confusion raised by reporting the lowest value as 0.0001 mg/L (Rossdale 1&2 treated minimum) when the detection limit was apparently 0.0003 mg/L.

Total chlorine residuals averaged only 0.26 mg/L for the Clareview reservoir. This is below the minimum of 1 mg/L required by the license and indicates a continuing problem with a chlorine demand occurring at this reservoir. The Local Board of Health collected 27.5% more samples (a total of 2296 samples) from the distribution system for bacteriology analysis by the Provincial Laboratory of Public Health than specified by the guidelines[10].

Odor described as musty was reported for up to 15 days at Rossdale and 10 days at Smith. Complaint statistics were down to a total of 220 from 300 in 1981. The distribution of complaints supports the assessment that odor was not as intense in the spring as the previous year.

Overall, reporting in 1982 was substantially improved over the dismal 1981 report. However, there was still an absence of documentation for clear followup action to eliminate problems which were causing license limits to be exceeded.

1983. The improvement in reporting and corresponding performance in 1982 continued according to the 1983 Annual Report[37]. The major concern in 1983 was the outbreak of Giardia. The possibility that water from the Rossdale plant may have been involved was summarized in the report[37] as follows: " A subsequent epidemiological study, conducted by the Edmonton Local Board of Health, failed to establish a cause, while noting clustering of cases around the City's core. The interpretation that the water supply from the water treatment plant serving that area was the cause of the spread of infection, could not be substantiated."

The report[37] goes on to note that implementation of chlorine dioxide on a year round basis was pursued following this incident, as a conservative measure to increase barriers to Giardia penetration of the plant. Because there is no explicit Giardia parameter for the license, this subject will not be pursued further here, rather it will be discussed in section 3.6.

Control of fluoride values continued to be a problem. A total of 6 values ranging from 1.25 to 1.4 mg/L were over the license limit of 1.2 mg/L for a daily maximum. Again no discussion was provided to indicate any intended action to bring this continuing problem under control.

Only one day was listed for an offensive odor, described as musty. These observations do not account for the taste and odour complaint statistics, which are down from previous years but are still clustered in February, March, April and October. These are months which experienced clustering of taste and odor complaints in one or other of the previous two years (Table 6.18). These statistics suggest that: taste and odor monitoring at the plant was insensitive to the problems perceived by some consumers, the problems arise in the distribution system, or the complaints are all spurious. The last possibility does not seem reasonable.

Once again, the phenols limit was exceeded 9 times with maximum values reaching .005 mg/L. These occurrences were also not correlated with taste and odor events as noted in 1982.

One value for antimony exceeded the objective level of 0.0002 according to the summary in Table 6.14. That is not a significant concern for the reasons given in discussing 1982. However, the reporting in Table 6.1 does not reflect the observations of Table 6.14. The maximum value for Rossdale 1&2 treated is left blank and several values are reported as <0.00 mg/L, a meaningless designation. This line of Table 6.14 was apparently not accurately completed and proofread.

A total of 15 violations on pH were noted with the explanation: "Obsolescence of plant. Scale-forming potential increased only slightly" Because 12 of the 15 violations occurred at the new E.L.Smith plant, the obsolescence explanation is inappropriate.

A new scheme of reporting turbidity based upon an average of 24 (Rossdale) or 12 (Smith) individual samples over 24 hours contributed to keeping maximum values below 3.3 NTU. Intermittent values would be expected to reach much higher than this and would raise the concerns about Giardia breakthrough expressed previously.

Reservoir chlorine residuals continued to be a problem with Clareview averaging 0.44 mg/L, Londonderry 0.93 mg/L and Rosslyn 0.93 mg/L compared with the license requirement of 1.0 mg/L. The Local Board of Health collected 68% more samples (a total of 3031) from the distribution system for bacteriology analysis by the Provincial Laboratory of Public Health than specified by the guidelines.[10]

A special survey was conducted in 1983 to evaluate asbestos fibre occurrence in raw and treated drinking water for both plants and for the distribution system. Fibres were only rarely encountered and then mainly in the raw water. The results suggest that asbestos fibres provide no cause for concern in the Edmonton system. This finding is consistent with the expectations expressed by Dr. Logsdon (Logsdon report, this Volume).

Overall, 1983 performance and reporting was similar to 1982. Serious health related violations were not apparent, but the chronic poor control of fluoride levels called for some specific remedial response which was not evident.

1984. The trend towards more detailed documentation and improved performance relative to license limits continued with the 1984 Annual Report.[30] In fact, the total amount of information contained in the annual report has become cumbersome and some duplication existed in the narrative sections. These are minor problems compared with the awful report of 1981, but room exists for some overview consolidation by senior management to reduce inconsistencies between different sections describing the same events.

A minor example in 1984 was a discrepancy between the plant operations and quality control sections. On page 10, plant operations mention a sudden increase in taste and odour complaints on August 27 and 28 at Rossdale caused by storm-related street runoff. On page 47, the summary of complaint statistics does not show any notable increase in quality complaints in August but notes as explanation for a high number of complaints in September that: "Lots of taste and odor complaints received due to street runoff on September 27 and 28th." Likewise, Page 11 in the operations discussion provides a summary of spill events and refers the reader to Table 5.17 which repeats this summary with data to indicate costs incurred by extra monitoring. The two lists are consistent except that the former mentions a gasoline spill on April 27 which is missing from Table 5.17.

The total number of violations from license limits quoted was 23 (not counting 257 violations for temperature). This included 5 for antimony which ranged from 0.003 to 0.008 mg/L. Earlier comments about limited significance of these values apply here and strictly speaking these values do not constitute violations of the license.

Fluoride continued to be a problem with a maximum value of 1.37 mg/L being reached among 13 daily violations, most of which were on low side (i.e. less than 0.9 mg/L) rather than excessive values. This represents a long history of consistent problems with fluoride control. No specific commentary about the need for remedial action was apparent.

A total of 4 pH violations (above 8.5) were reported and the same commentary about obsolescence of plant was provided. In this case, 2 of these violations occurred at the new E.L. Smith plant.

The maximum value reported for mercury in treated water at Smith was 0.0011 mg/L (Table 5.1) which constitutes a minor violation because the license limit is 0.001 mg/L. This value, which would have little significance unless it re-occurred, was overlooked in preparing the summary of violations (Table 5.13).

Previous problems with maintaining chlorine residuals in reservoirs were brought under control and levels were maintained at the license requirements. The Local Board of Health collected 53.6% more samples (a total of 2766) from the distribution system for bacteriology analysis by the Provincial Laboratory of Public Health than specified by the guidelines.[10]

Because free chlorine is not used at Edmonton, trihalomethanes have not been a problem relative to the license requirement. However, reporting in 1984 showed values to be consistently below a detection limit of 0.001 mg/L. Such values are difficult to believe and are not supported by the Alberta Environment treated water survey data for 1984.[72] That data shows values in the range from 0.0004 to 0.011 mg/L, with most values above 0.001 mg/L, based on samples from the distribution system. Either set of values are well below the license limit of 0.350 mg/L but the consistent reporting of non-detectable values by the Edmonton lab raises concerns about the quality of their analyses for this parameter.

The severity of taste and odor events was apparently decreased from 1983, particularly during the spring runoff. However, one incident of oil contamination affecting Rossdale water in September was apparent (suspected from urban runoff) with a rise in consumer complaints. These were not reflected in the plant summary for taste or odor suggesting that in-plant monitoring was not effective in documenting the sensory quality of the water. This contamination, mentioned in section 2.6.1.1, was also clearly documented by chemical analysis so it cannot, in retrospect, be dismissed as an artifact resulting from over-sensitive consumers.

Finally, chlorine dioxide yields were reported as varying from 23% to 146%. Because the generator yield cannot be greater than 100% a clear anomaly existed. The low yields, if accurate, would have indicated substantial waste of the feed chemical, sodium chlorite, and the likelihood that chlorite, which will be discussed in more detail in section 3.6, was being added to the water with no associated benefit.

Overall, the quality of this report and the exteme values of treated water quality described have continued to improve from the low point of the 1981 report. However, the instances noted indicate there continues to be room for improvement in proofreading and cross-checking of information to assure that an accurate record has been presented. There are also indications of areas where in-plant monitoring is suspect and verification by increased analytical quality control is warranted.

1985. The annual report for 1985 is not available at the time of this report (June, 1986) so recourse will be made to the monthly reports for 1985. The year will be discussed in two parts January to June[51-56] and July to December.[57-62]

January to June, 1985. Taste and odor were major issues in the spring of 1985. The March report notes that hydrocarbons were experienced at Rossdale at the end of February. That event will be discussed in more detail in section 3.5.1. However, the February events were overshadowed by public response to taste and odor problems starting about March 20. A total of 106 complaints were received, with most coming between March 20 and 22. This period coincided with a very high demand for chlorine dioxide apparent in late March and early April. Plant records described the odor as musty, but a review of the complaints file indicated descriptors including musty or stale but ranging from bleachy to kerosene.

The problem was explained in the report[53] as: "further complaints came from the E.L. Smith supply zone as a result of sudden elevation of natural compounds (humates) which, at first, were incompletely oxidized." Raw water total organic carbon levels reached 10 mg/L in April. Complete oxidation of such high levels of raw organic carbon, even with very high doses of chlorine dioxide, should not be expected. However, formation of a wide variety of byproduct organic chemicals would be expected.

High doses of chlorine dioxide were used in March and April. These reached 2.8 mg/L at Rossdale in April. Doses at Smith are unclear because of a possible error in plotting of Figure 2.10. Taking the figure legend as it stands in the report[54] the doses peaked at 3.7 mg/L. At the same time, residual chlorine dioxide is reported at near zero. Because the license did not specify controls on chlorine dioxide or chlorite, chlorite residuals at the times of high dosage were not reported. However, experience elsewhere suggests that chlorite appearing at levels of 40 to 60% of chlorine dioxide dosage are usually expected.[75]

Phenols were reported at various times in April, May and June at levels up to 0.006 mg/L compared with the license limit of 0.002 mg/L. These levels are as high as some levels reported in previous years as being associated with taste and odor. Such levels were apparently not associated with taste or odor in 1985. This casts some doubt on the importance of the phenols (reported by the standard colorimetric method) as a significant cause of taste or odor. Figure 1.9 which had been providing a useful summary of complaint statistics in comparison with previous years was dropped with the May report[55] and was not reinstituted for the rest of the year or in 1986.

July-December 1985. This operating period was somewhat less eventful than the first half of the year. A set of samples of raw and treated water for all plants was sent to Cantest Laboratories for pesticides, herbicides and priority pollutant analysis. The results were consistent with the monthly Alberta Environment treated water surveys[71-74] in that only the commonly found phthalate, bis(2-ethylhexyl) phthalate (BEHP or DEHP) was found in any of the samples. This compound is not subject to a license limit. The significance of BEHP is discussed in the reports by the Canadian Center for Toxicology and Dr. Bull (this Volume).

Over the summer, problems seemed to be apparent with Rossdale plant 3. Chlorine dioxide dosage was erratic in July with values reaching 3 mg/L. This plant was shut down on July 21. The problem was described as an inability to maintain filter effluent residual chlorine dioxide above 0.10 mg/L. That presumably explains the high doses which were applied. Various experiments were run with the plant running to waste. Plant 3 was apparently placed back in service on August 5 but the exact nature of the problem was not described.

September brought heavy rains resulting in high raw water colour and turbidity values. Chlorine dioxide demands at Smith as high as 3.6 mg/L are discussed although Figure 2.10 shows values peaking at 3.2 mg/L. Presumably the difference lies between short term peak demand in the former case versus the daily average in the latter. In either case, these represent very high chlorine dioxide doses. The doses were limited by capability of the generation equipment and total water production was reduced, accordingly. The problem was described as: "difficult runoff conditions, at which times it is imperative to feed sufficient ClO_2 to meet demand, i.e. to avoid the formation of chlorophenols."[59] That explanation does not provide a technically sound rationale for applying high chlorine dioxide doses. High residual chlorine dioxide values of 0.27 and 0.28 mg/L were reported for the Kaskitayo and Papaschase #2 reservoirs, respectively, in November.

Another question arising out of the application of chlorine dioxide in September, is why the demand should have been higher at Smith than at Rossdale. Generally, Rossdale will experience the same or higher organic loadings than Smith. Considering the reported colour values, which seemed to correlate with the shape of the demand curve, the loading at both plants appears identical. Referring back to April 1985, when high demands were experienced at both plants, there are only marginally higher color values reported for Smith than for Rossdale. Yet, the apparent chlorine dioxide demand was much higher at Smith. Finally, referring to the limited total organic carbon data for both periods, there is no significant difference between plants. Given all these observatons and the history of poor and inaccurate chlorine dioxide generator efficiencies, I cannot rule out the possibility of an apparent high demand at Smith arising, in part, from inaccurately measured generation. In this case, chorite would be added to the water partially or largely unconverted. Such an occurrence would mean that water was dosed with high values of chlorite, but with no associated benefit and a substantial waste of money (high demand daily chlorite consumptions over 500 kg/d at $4.50/kg).

In addition to the raw water problems, treated water was run to waste for 3 hours at Smith when organics were detected by gas chromatography. These were later attributed to a new sample pump which had contaminated the sample. However, the incident indicates that process monitoring for organics was being watched and appropriate responses were initiated from the results. That approach led to solution of another situation which could conceivably have been causing intermittent problems previously. On October 10, gas chromatography analyses identified that trace organics were present in natural gas boiler flue gases which had been used as a source of carbon dioxide for recarbonation. Once identified, this source was eliminated.

On October 29, the Smith plant was shut down for 2 hours because of the detection of a musty odor. No further action was necessary. Plant 3, at Rossdale, continued to exhibit problems. The report[60] noted: "Because of the detection of the penetration of the treatment process by viable bacteria, the chlorine residual was raised to 2.3 mg/L between the 12th and 22nd of October." This action apparently solved the problem at that time. However, in December plant 3 was shut down again because of high volatile organics as measured by gas chromatography. The plant summaries also indicated an anomaly with total organic carbon. Treated water from plant 3 had a higher concentration than raw water, 4.7 vs 3.8 mg/L. No explanation was apparent for this. Problems with detected total coliforms were again apparent with plant 3 in December and again resulted in increased chlorine dosage.

There were also anomalies with regard to cyanide. Treated water values were reported at 0.011 and 0.012 mg/L whereas raw water was only 0.002 mg/L. None of these values remotely approaches the license limit of 0.2 mg/L. However, the possibility of bulk chemical contamination needs to be investigated.

Quality control checks were instituted on lab technicians and operators in October. Results indicated some deficiencies, notably with color measurements. There seemed to be some reluctance on the part of some operators because not all samples requested were being returned.

Overall, this period indicated some worthwhile initiatives and good treatment performance even during very poor water conditions. The major concerns are the continuing problems which occur with Rossdale plant 3 and the manner in which chlorine dioxide was used. As mentioned before, the latter will be discussed in more detail in section 3.6.

January to May 1986.[63-67] The most recent time period reflects a continuation of the trend apparent throughout much of 1985, namely, good treatment performance with minimal variance from license requirements despite many challenging raw water conditions. In general, the monthly reports reflect early identification of problem conditions and sustained efforts toward achieving solutions.

Problems with adequate disinfection at plant 3 continued, leading to plant shutdown and superchlorination. In February,[64] backwash flowrates were found to be too high and a new backwash cycle was implemented with improved results. Higher total organic carbon values in plant 3 effluent than reported for raw influent was apparent again in February. This should be investigated to determine if it is only an anomaly of raw versus treated sampling times. If not, the source of organic carbon increase at plant 3 must be determined.

Increased cyanide values (0.004 treated vs <0.001 raw) were also noted in February. The April report refers to methods development which is underway for bulk chemicals analysis. This must be considered a priority given occurrences like the cyanide example, even if values in this case are well below license limits.

The quality control program with technicians and operators continued to be reported. Early in the year, return rates by operators were still unsatisfactory, but improvement occurred by May. Performance was generally good, but given the number of trials being done, unacceptable results should become exceptional rather than occasional.

The Rossdale plants survived a period of intense urban runoff at the end of February without allowing any taste or odor breakthrough. On March 1, rural runoff hit and brought taste and odor problems to both Smith and Rossdale. These events will be considered in more detail in section 3.5.2.

Alberta Environment requested reporting on chlorite during February.[64] Although the measurements which could be used for this purpose were available, there was a time lag necessary to back-calculate results. When the high organic loading in the river hit on March 1, dosage of chlorine dioxide was increased at all plants in an attempt to meet demand. Peak dosages hit 2.75 mg/L at Smith. In response, Alberta Environment specified that dosage of chlorine dioxide be limited to the extent necessary to keep the combination of chlorine dioxide plus chlorite (called total oxidants) below 0.5 mg/L. At Smith, the chlorine dioxide application point was moved to pre-softening from the previous raw water position. This resulted in an immediate drop in chlorine dioxide demand which made the maintenance of some chlorine dioxide residual possible within the total oxidants restriction. The benefit of reduced chlorine dioxide demand arising from moving the point of application was still apparent in April. Comparative dosage levels at Smith were 0.3 to 0.4 mg/L versus 0.5 to 0.9 mg/L at Rossdale.

The April report[66] also notes that the method used by operators for chlorine dioxide measurement includes measurement of chloramines and thus is an overestimate. The report[66] states that: "An error in this test is acceptable, since an error is on the side of safety." This interpretation neglects the two-sided nature of the chlorine dioxide issue. While over-estimation of chlorine dioxide may provide greater safety from overdosing, the error provides correspondingly less safety if relying on chlorine dioxide as a primary disinfectant.

Differences in fluoride measurements between the Alberta Environmental Center and the Edmonton Water Labs were discussed.[65] Some difference could be attributed to sample type (grab versus composite) although fluoride levels should not fluctuate much under good process control conditons. Continued cooperative work was proposed to rectify the differences.

Finally, some transcription errors apparently crept back into the tabulation of quality analyses for May.[67] Values of <0.76 mg/L for arsenic, 0.35 to 0.54 mg/L for mercury and <0.75 mg/L for selenium were reported. These values must have all been intended to be reported as ug/L and the appropriate adjustment of 1000 missed in entering them in the Table 2.2.1.b. Because the May report is essentially "hot off the press", there has not been time for corrections arising from management review of the report.

Summary. The performance of the Edmonton Water Treatment Plants based upon review of their Annual and Monthly reports can be categorized into general issues and specific cases of license violations.

Dealing first with license violations, there was a distinct trend toward a decrease in the number, significance and degree of license violations over the review period (1980 to May 1986). Only temperature violations, which are largely beyond the control of plant personnel, and odor complaints escaped this trend. Occasional very high metals violations which occurred in the early 1980's have not been noted in recent years (assuming the case for May 1986 proves to be a transcription error). Frequent fluoride and occasional pH, color and turbidity excursions have been largely eliminated. Marginal phenols violations still occur but with less frequency and severity than evident in the early 1980's. Phenols violations have not been correlated with taste and odor complaints. Problems with maintaining licensed levels of chlorine residual in distribution reservoirs appear to have been overcome.

The current practice of trying to meet objective levels which are below the license limits for several parameters is commendable and is likely beneficial in assuring good license compliance.

In conclusion, the current level of compliance with the Clean Water Act licenses, based upon Edmonton's records, is excellent, particulary in contrast to the blemished record only a few years ago. A review of Edmonton's performance based upon independent, provincial data is provided in section 3.2.2.

Given the extensive information base contained in the City's reports, several general issues arise which warrant discussion. Although, the quality of proof-reading of reports has improved dramatically since the low point of 1981, there is still room for improvement. Having taken the commendable action of providing a publicly accessible written record of their performance, the City has a duty to keep it as accurate as possible.

In the review, several cases are noted where explanations of license violations or variances from objectives are not thoughtfully explained. Because good performance has substantially reduced the number of cases requiring discussion, more evidence of thoughtful analysis of remaining problems and possible solutions would be useful. Likewise, clear explanations of followup actions are often but not always provided. One justification for devoting the resources currently committed to preparing these detailed reports would be the value of providing a history of problem solving to assist with operations during future personnel turnover.

The case of traces of cyanide showing in treated water and not in raw is excellent justification for re-instating bulk chemicals analysis which was abandoned in 1980.[68] Progress on the quality control program is encouraging but it must be maintained as a high priority. Further discussion of this is provided by the Kratochvil report (this Volume).

Performance of plant 3 at Rossdale continues to be a problem. Although task studies have been directed at this plant before, upgrading the performance and reliability of this plant should be an early priority of an in-house process development team. This concept, introduced by the Logsdon report, and supported by several others, will be discussed further in the conclusions to this report.

Finally, although chlorine dioxide controls were not explicitly part of the license, a review of practice suggests that strict controls are necessary as part of future license conditions. The rationale for using chlorine dioxide seems to have been mixed between attempting to oxidize odor causing substances and a perceived need to maintain a residual for protection against Giardia penetration. The latter has merit, the former is open to question. This will be pursued in more detail in Section 3.6

In either case, the dosage of chlorine dioxide to meet deamnd, limited only by what the equipment will allow, is not acceptable practice. That position is recognized in the new requirement by Alberta Environment that total oxidants (chlorine dioxide plus chlorite) shall not exceed 0.5 mg/L. That limit must still be regarded as an interim measure considering the concerns raised about chlorite in the report of Dr. Bull (this Volume). A rationale for evolving future chlorine dioxide practice is presented in section 3.6.2.

3.2.2 Review of Treated Water Surveys (1984,1985)

Alberta Environment has conducted treated waters surveys on Alberta's major communities since 1978. The scope of these studies has steadily increased. The 1984 and 1985 surveys[72,73] involved samples collected once per month at taps in the distribution system for each of the Smith and Rossdale plants. A total of 204 parameters were analyzed for each sample. The detailed results for surveys from 1983[71] to partial results for 1986[74] are included in tabular form in Appendix E.

Results for Edmonton Rossdale and Edmonton Smith for 1984 and 1985 were mainly evaluated for health-related parameters from the Canadian Drinking Water Guidelines[10] by plotting monthly values against license limits. These are provided for Rossdale in Figures 8.1 to 8.36 and for Smith in Figures 9.1 to 9.36.

Review of Rossdale results indicates that most parameters are present at insignificant levels relative to the guidelines. The only exception was iron which peaked at about 80% of the license limit in July, 1984. The iron limit is specified for aesthetic rather than health reasons, but should not even pose an aesthetic problem for an individual sample at that level. Other metals with measurable values peaked at fractions of the license limit of: 40% (cadmium), 20% (chromium) and 10% (copper).

Review of Smith results indicates that most parameters are also present at levels insignificant relative to the guidelines. The maximum value was a single observation for cadmium at 60% of the license limit. Chromium and mercury peaked at 20% and copper at 10% of their license limits.

These results are important in four ways.

1.They provide important, independent corroboration that Edmonton's water is meeting health-related guideline values.

2.Samples are collected more frequently (once per month) than City sampling for parameters such as the metals.

Fig. 8.1. **1984 Treated Water - Rossdale**

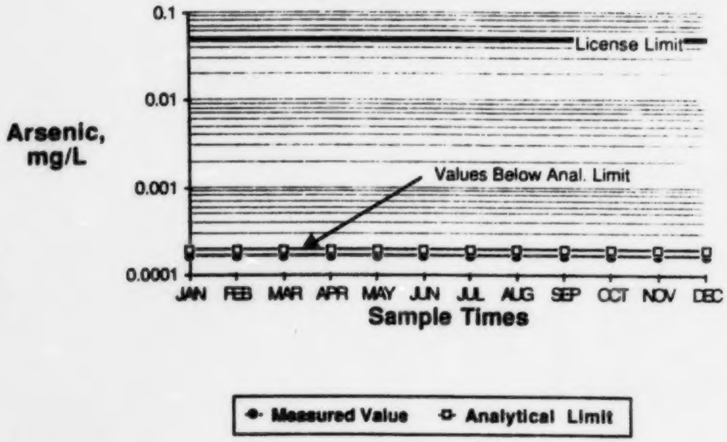


Fig. 8.2.

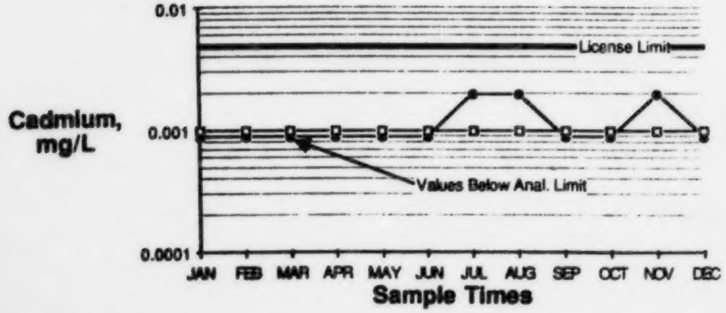


Fig. 9.1. **1984 Treated Water - E.L. Smith**

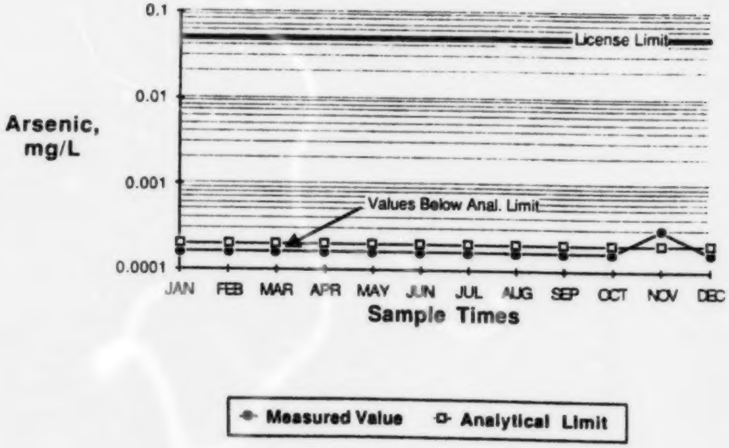


Fig. 9.2.

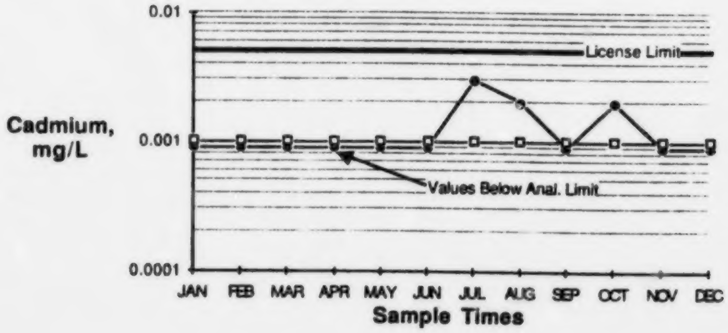


Fig. 8.3. 1984 - Treated Water- Rossdale

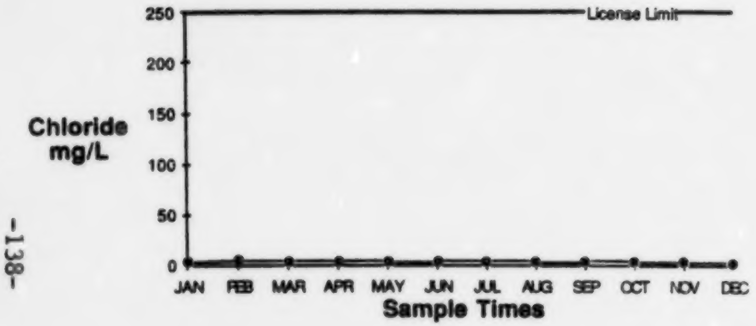


Fig. 8.4.

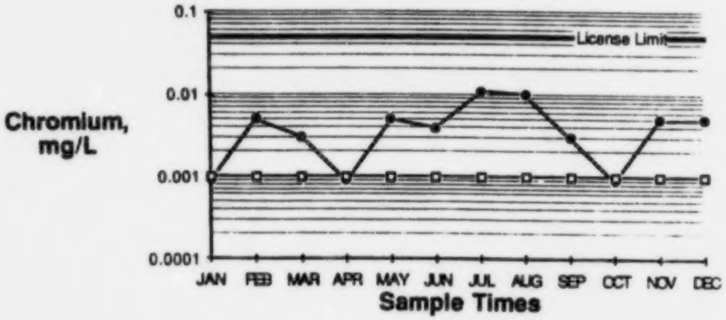


Fig. 9.3. 1984 - Treated Water- E.L. Smith

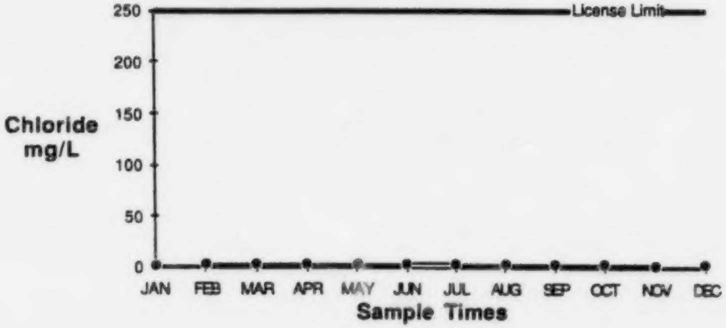


Fig. 9.4.

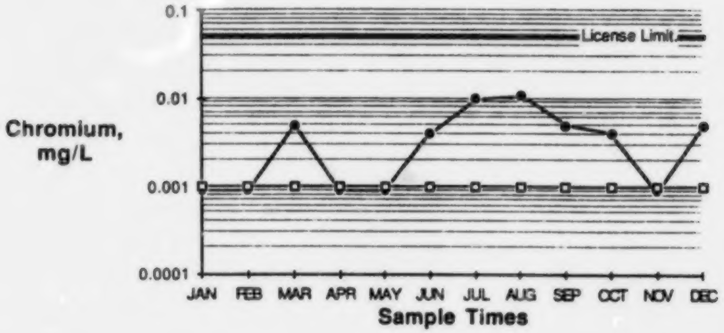


Fig. 8.5. 1984 Treated Water - Rossdale

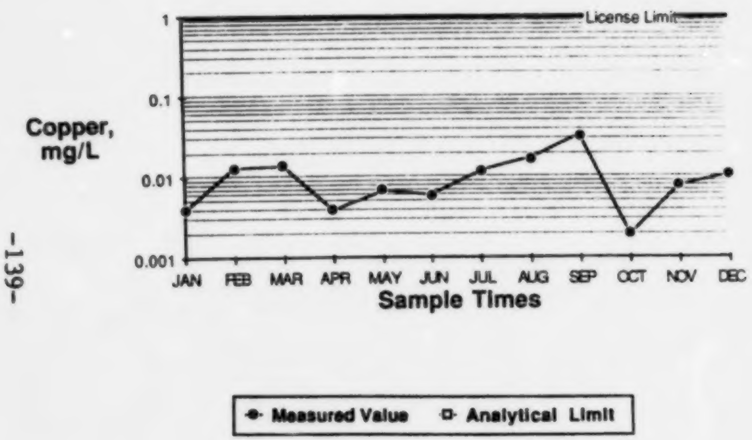
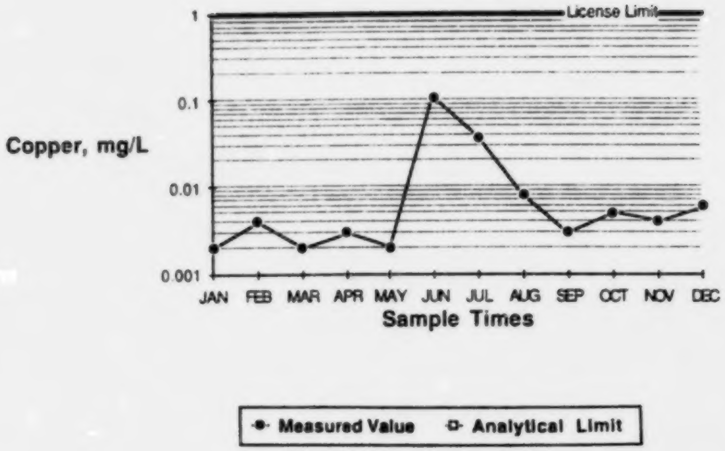


Fig. 9.5. 1984 Treated Water - E.L. Smith



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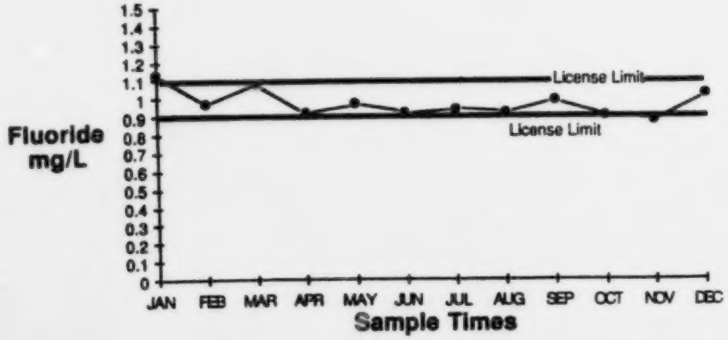


Fig. 9.6.

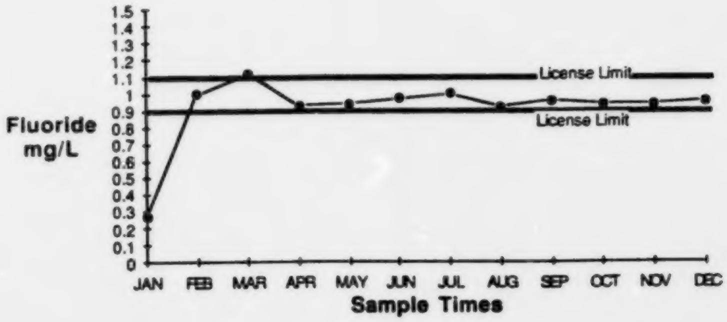
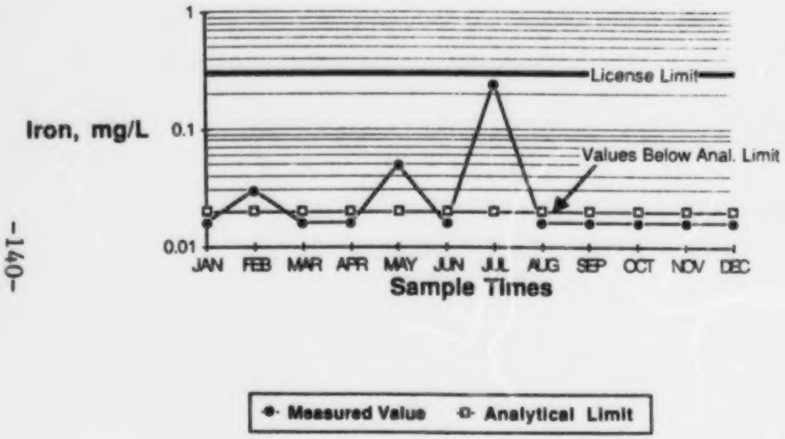


Fig. 8.7. **1984 Treated Water - Rossdale**



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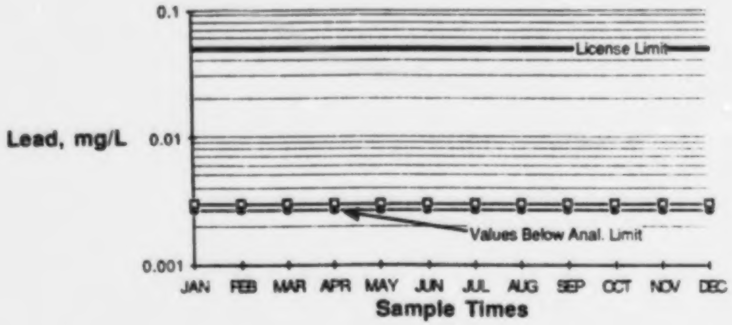
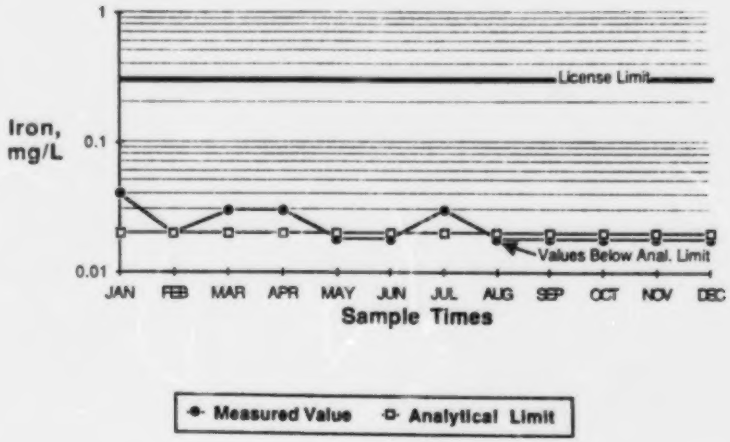


Fig. 9.7. **1984 Treated Water - E.L. Smith**



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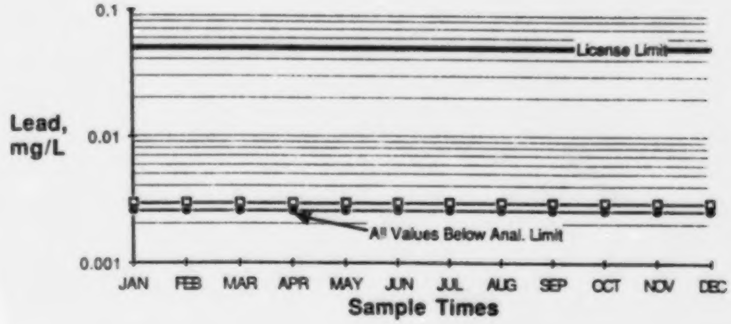
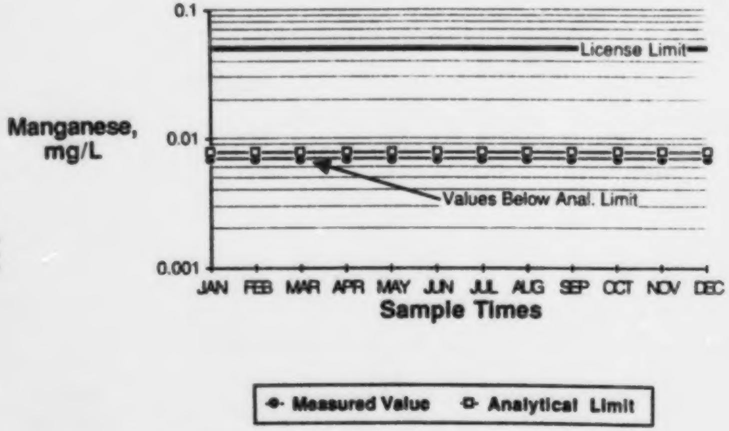


Fig. 8.9. 1984 Treated Water - Rossdale



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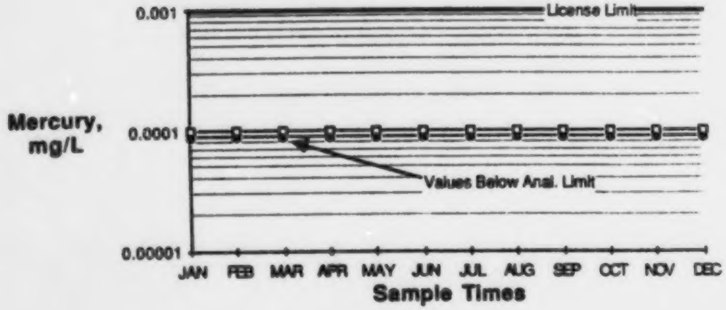


Fig. 9.9. 1984 Treated Water - E.L. Smith

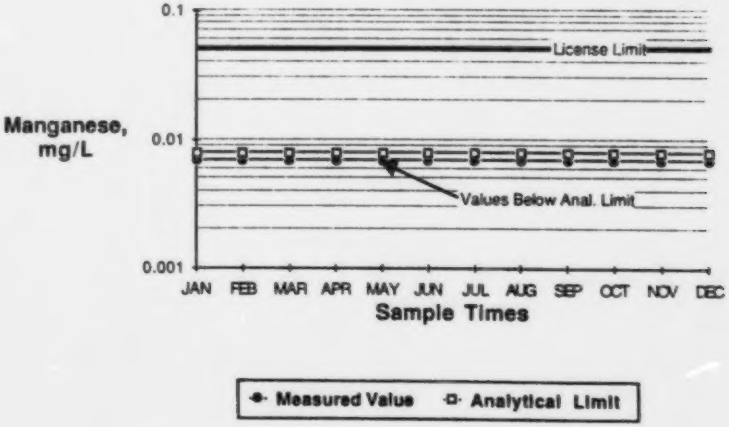


Fig. 9.10.

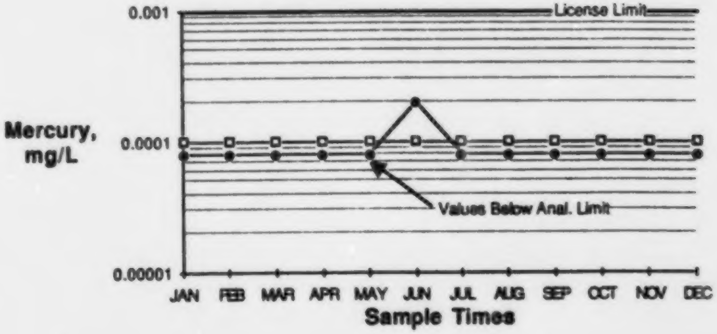


Fig. 8.11. **1984 Treated Water - Rossdale**

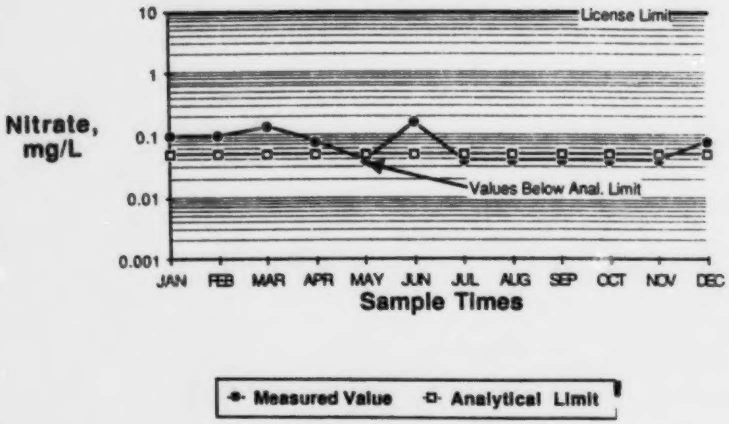


Fig. 8.12.

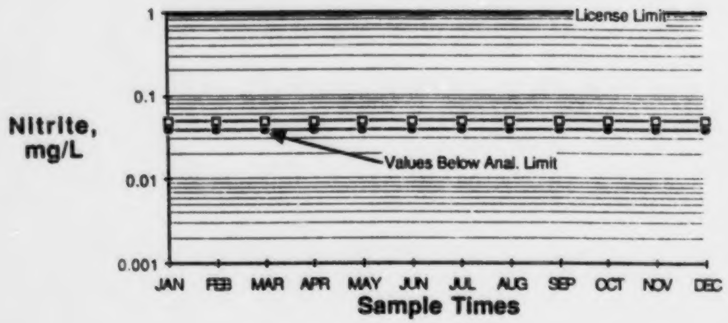


Fig. 9.11. **1984 Treated Water - Smith**

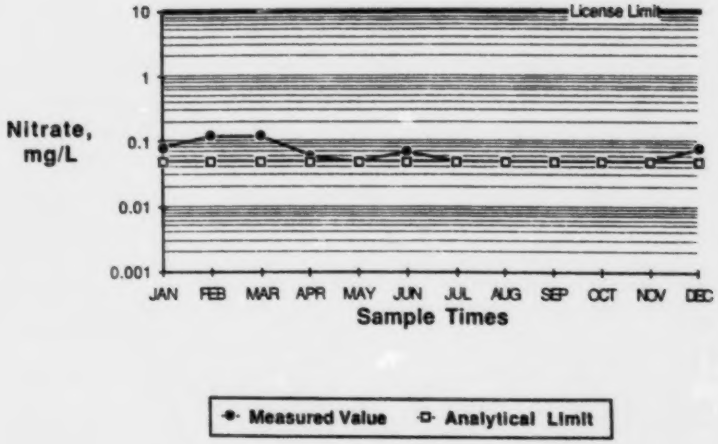


Fig. 9.12.

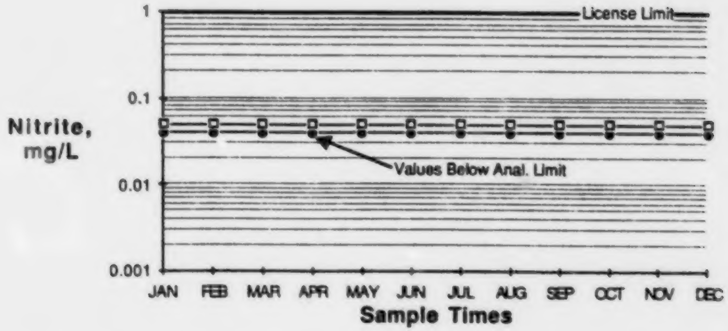
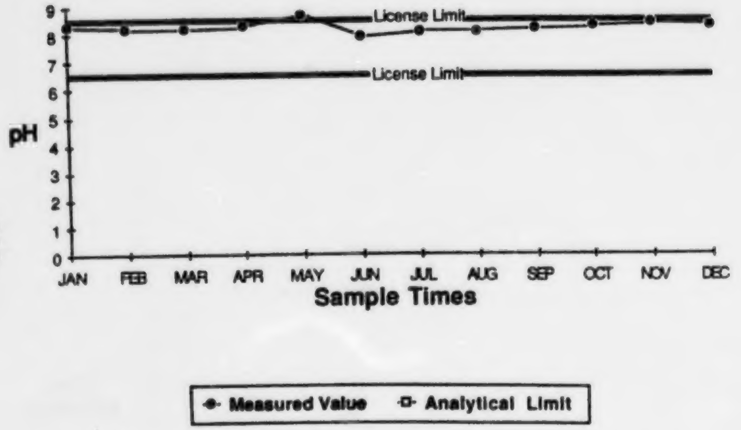


Fig. 8.13. 1984 - Treated Water-Rossdale



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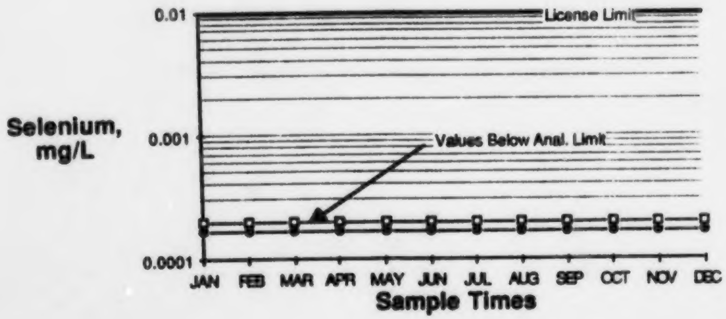


Fig. 9.13. 1984 - Treated Water- E.L. Smith

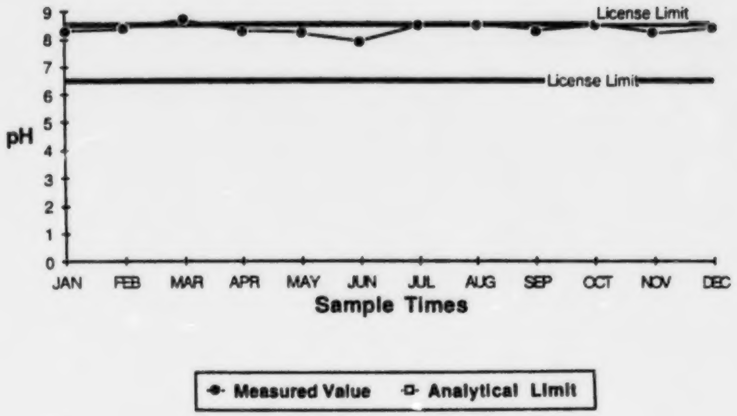


Fig. 9.14. 1984 Treated Water - E.L. Smith

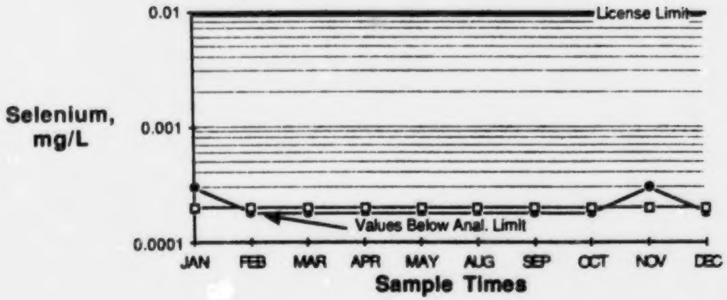
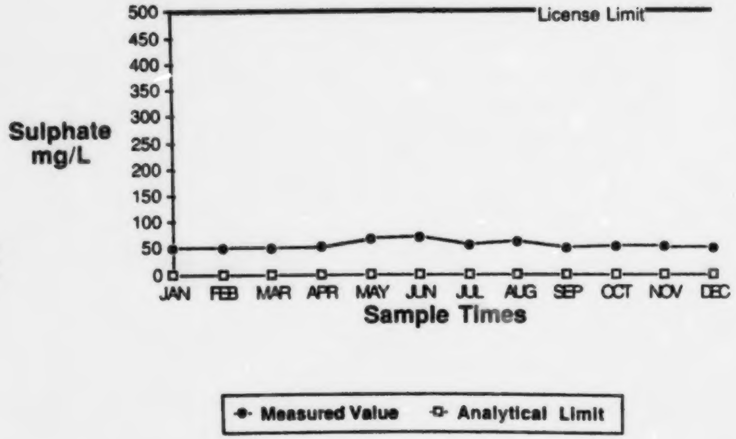


Fig. 8.15. **1984 - Treated Water-Rossdale**



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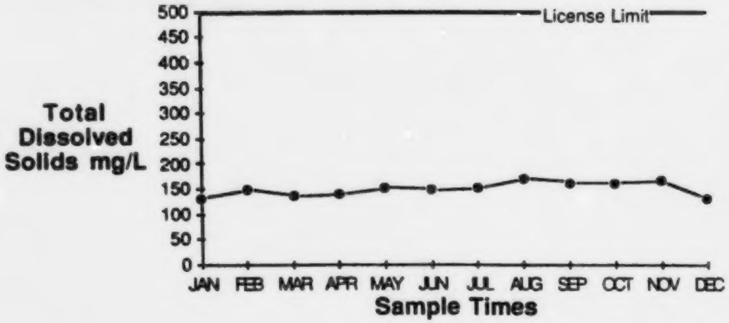
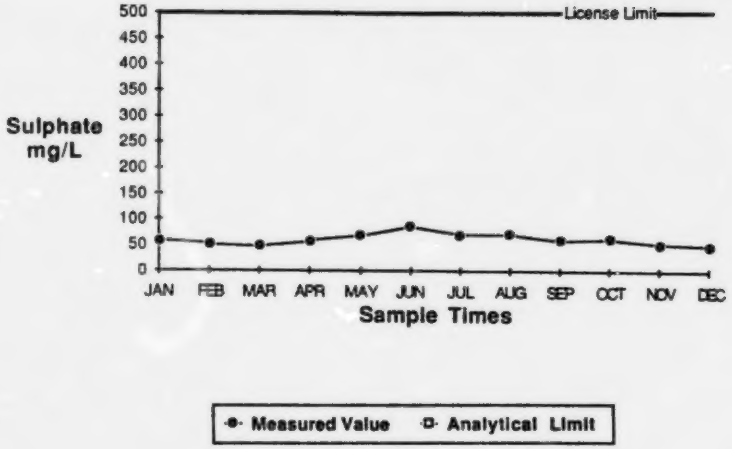


Fig. 9.15. **1984 - Treated Water- E.L. Smith**



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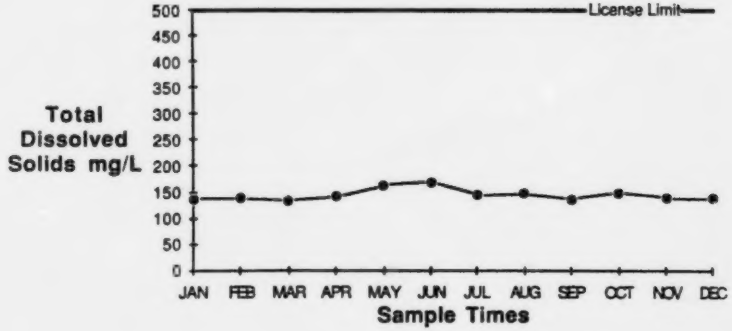
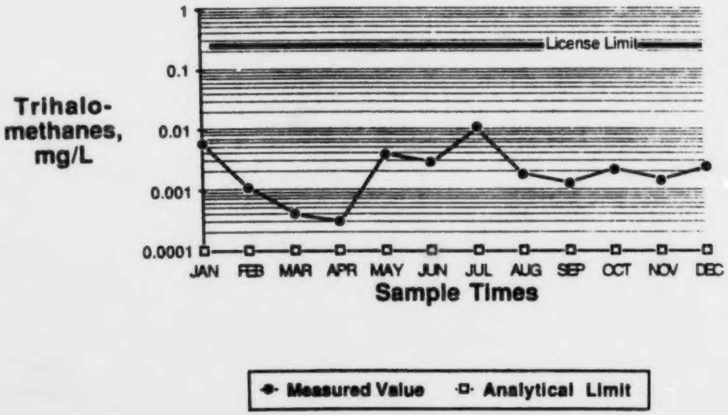


Fig. 8.17. **1984 Treated Water - Rossdale**



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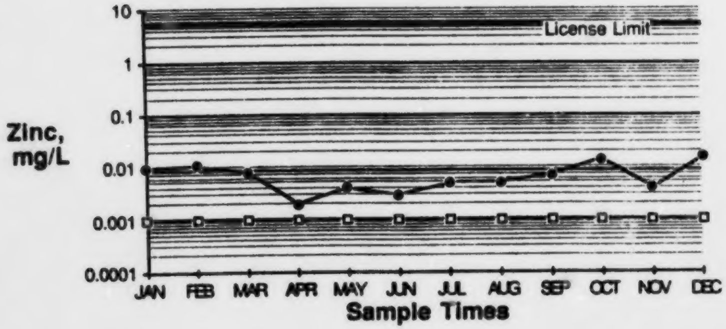
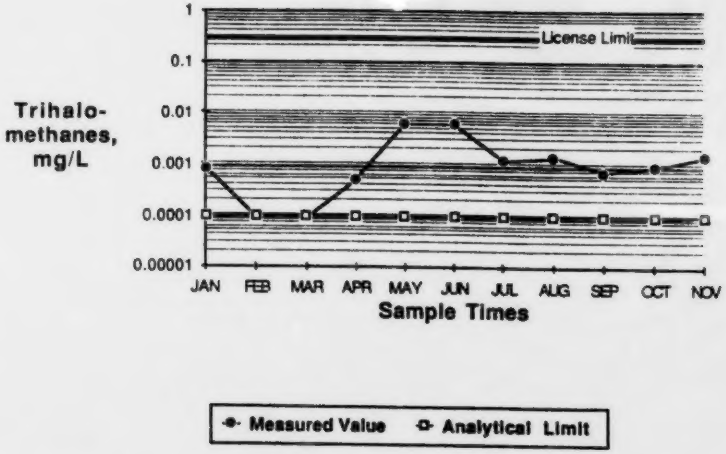


Fig. 9.17. **1984 Treated Water - E.L. Smith**



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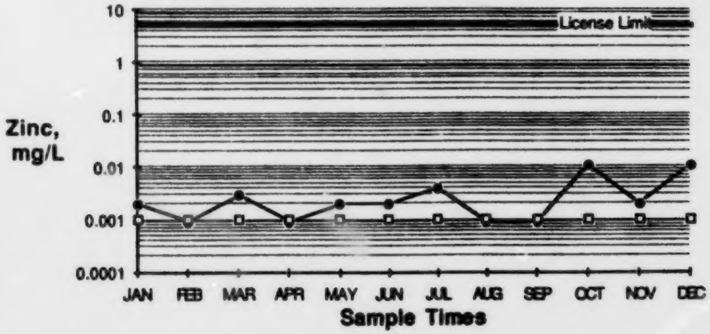
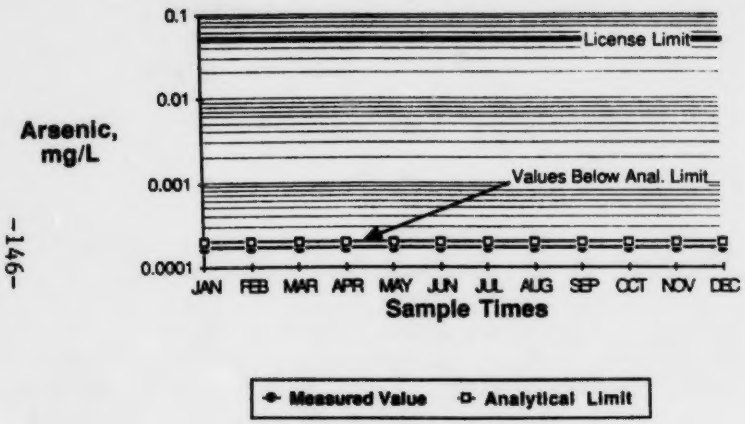


Fig. 8.19. **1985 Treated Water - Rossdale**



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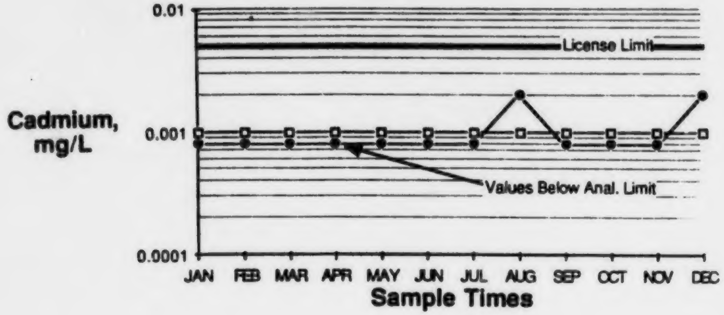


Fig. 9.19. **1985 Treated Water - E.L. Smith**

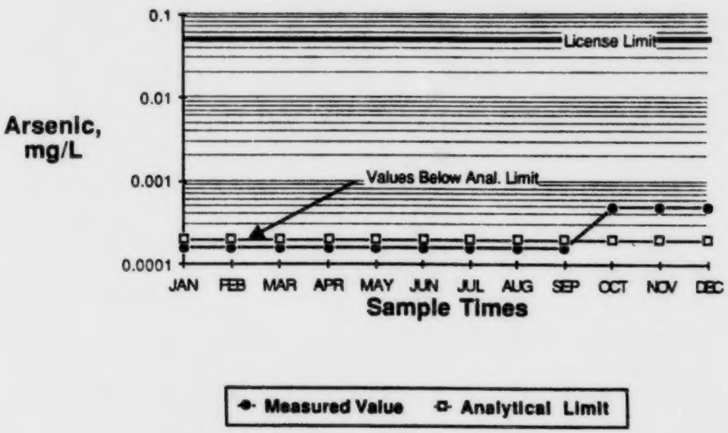


Fig. 9.20.

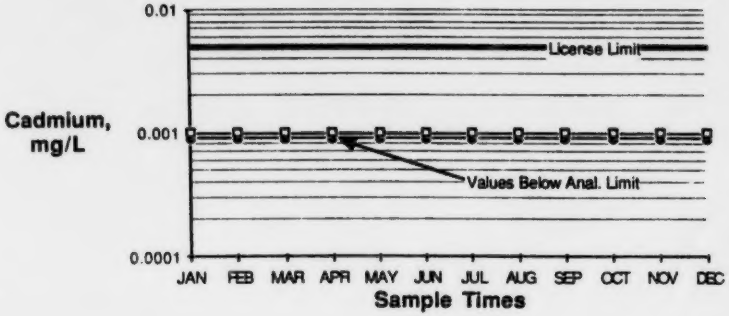
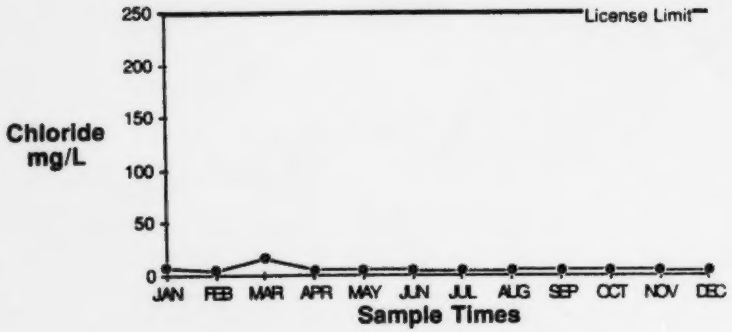


Fig. 8.21. **1985 - Treated Water- Rossdale**



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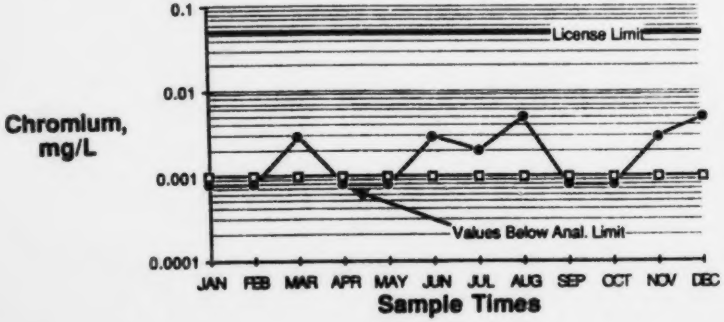


Fig. 9.21. **1985 - Treated Water- E.L. Smith**

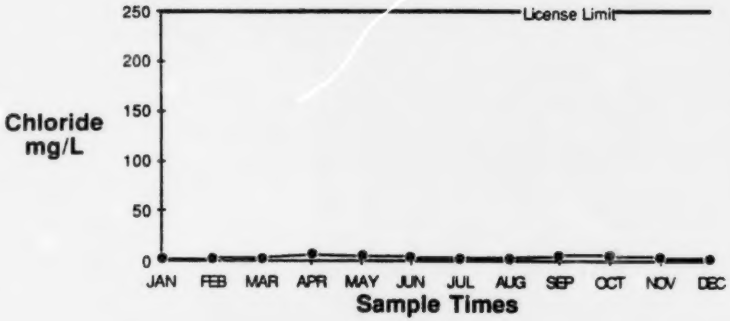


Fig. 9.22.

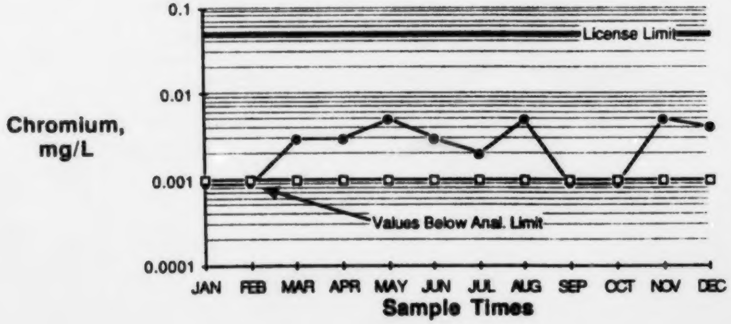
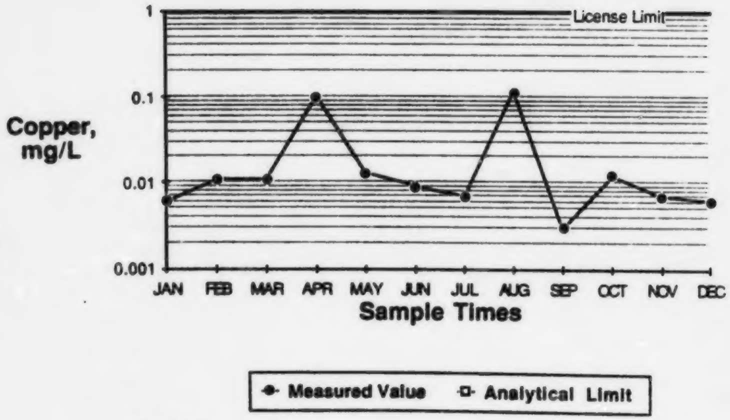


Fig. 8.23. **1985 Treated Water - Rossdale**



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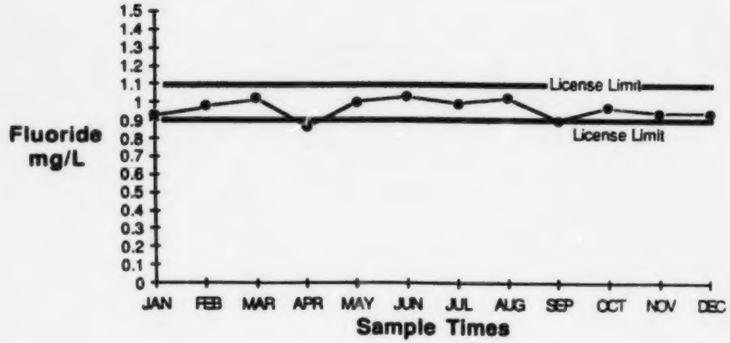


Fig. 9.23. **1985 Treated Water - E.L. Smith**

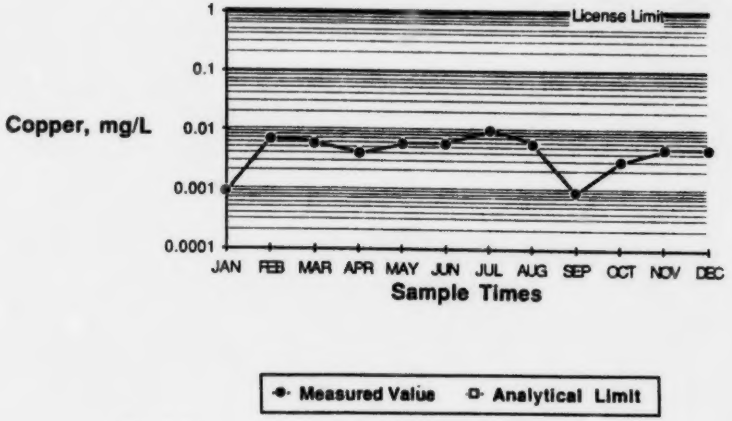


Fig. 9.24.

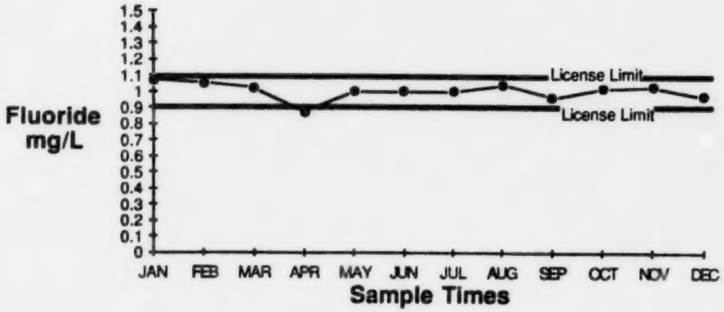
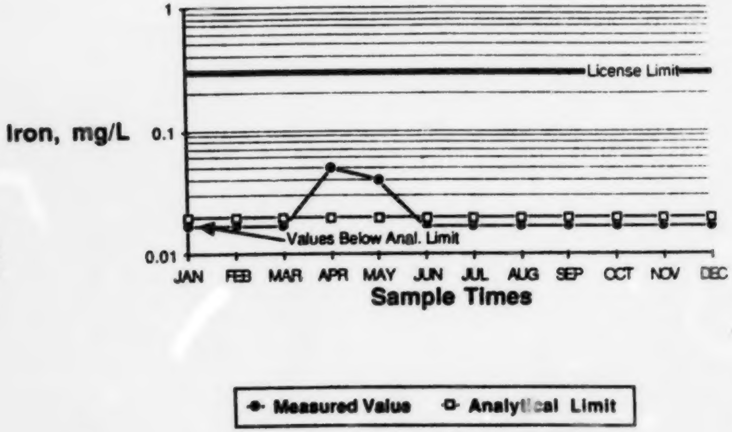


Fig. 8.25. 1985 Treated Water - Rossdale



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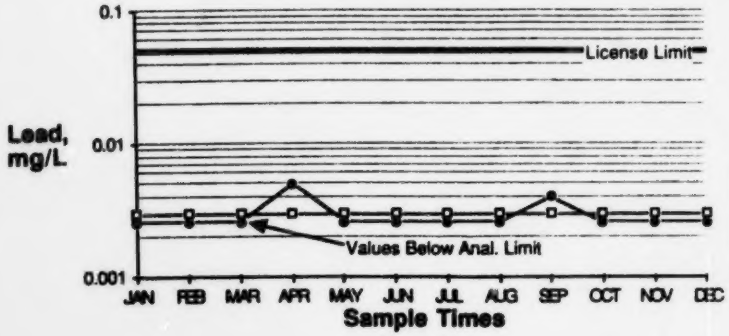


Fig. 9.25. 1985 Treated Water - E.L. Smith

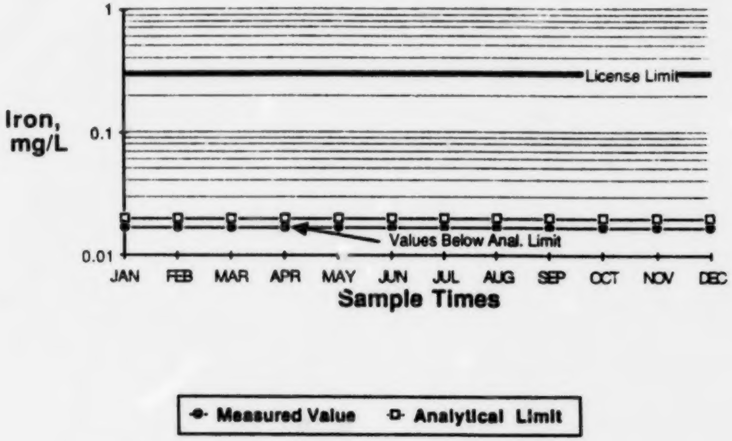


Fig. 9.26.

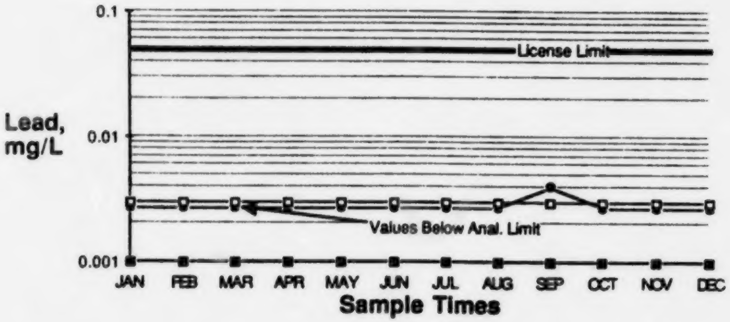


Fig. 8.27. **1985 Treated Water - Rossdale**

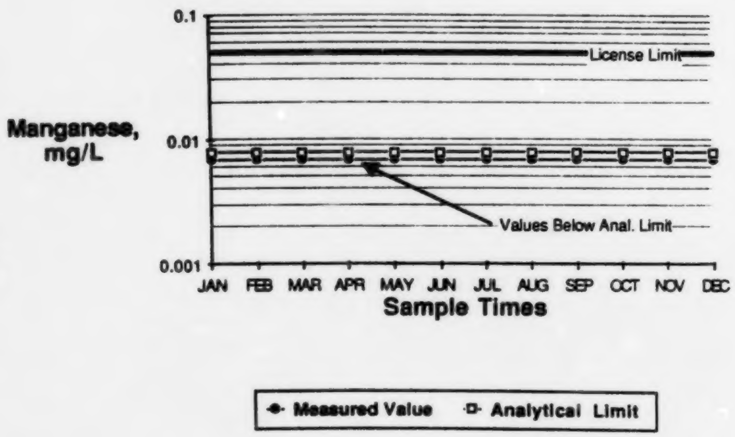


Fig. 8.28.

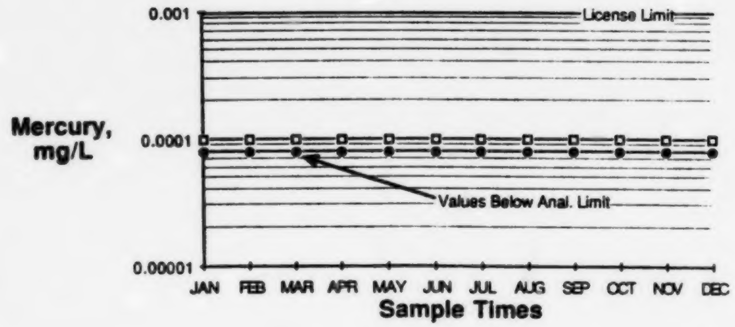


Fig. 9.27. **1985 Treated Water - E.L. Smith**

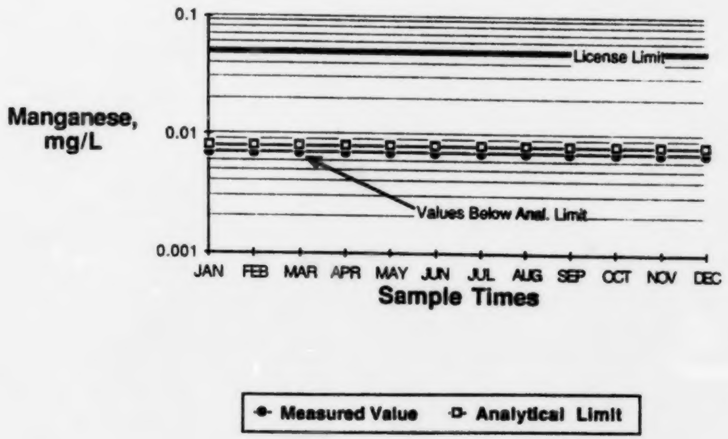


Fig. 9.28.

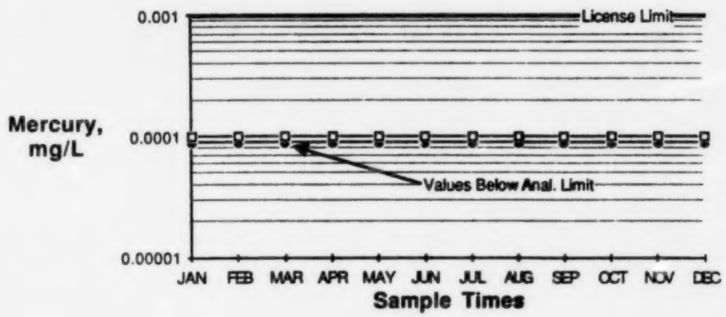
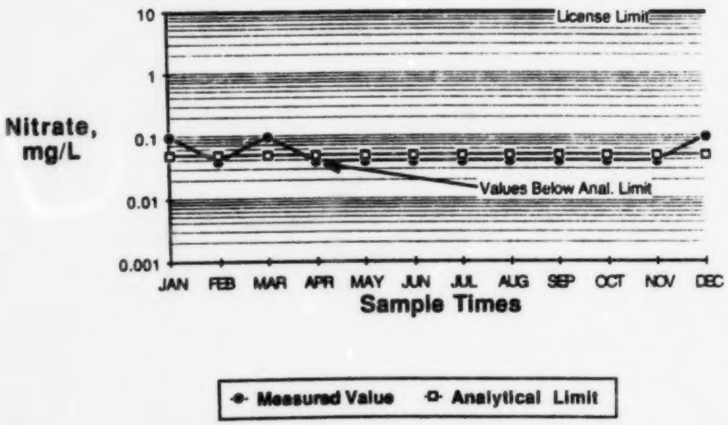


Fig. 8.29. 1985 Treated Water - Rossdale



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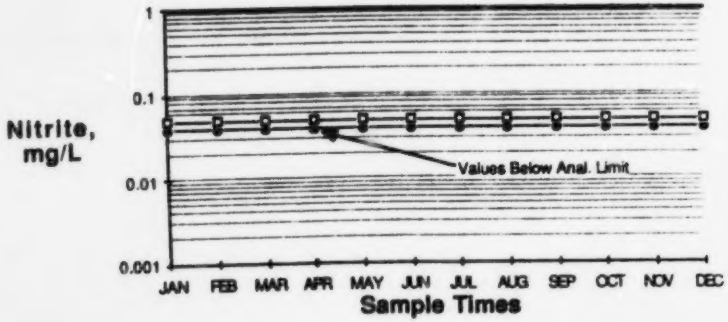


Fig. 9.29 1985 Treated Water - Smith

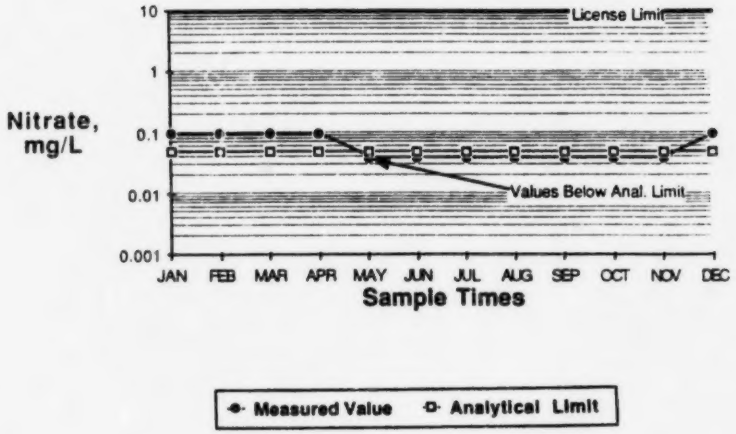


Fig. 9.30.

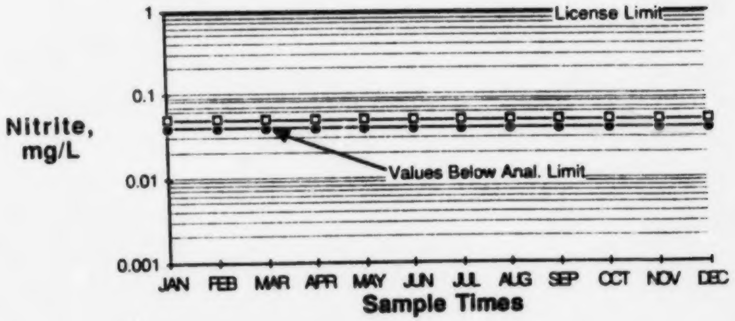
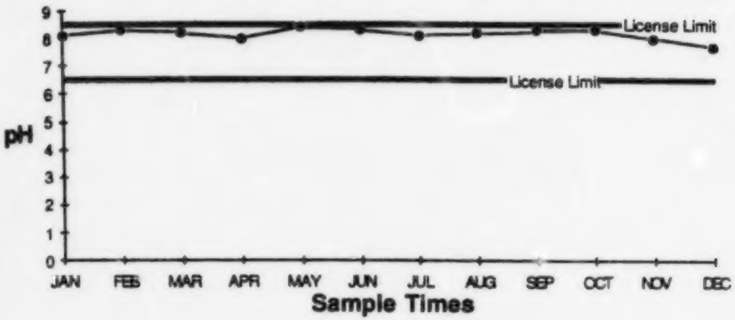


Fig. 8.31. **1985 - Treated Water-Rossdale**



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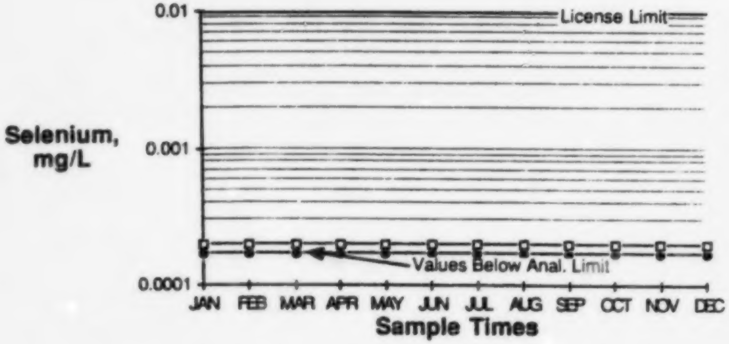
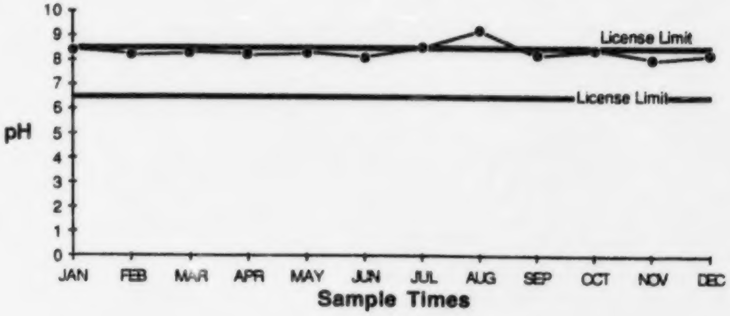


Fig. 9.31. **1985 - Treated Water- E.L. Smith**



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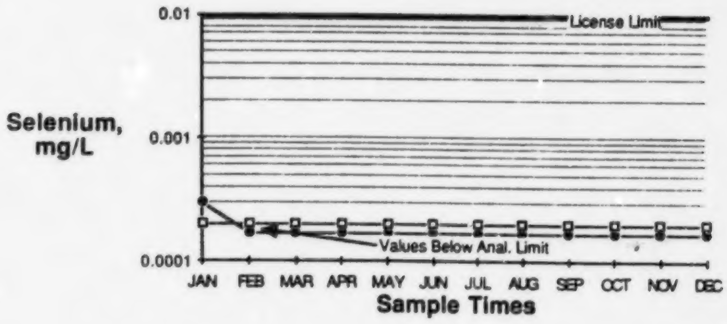
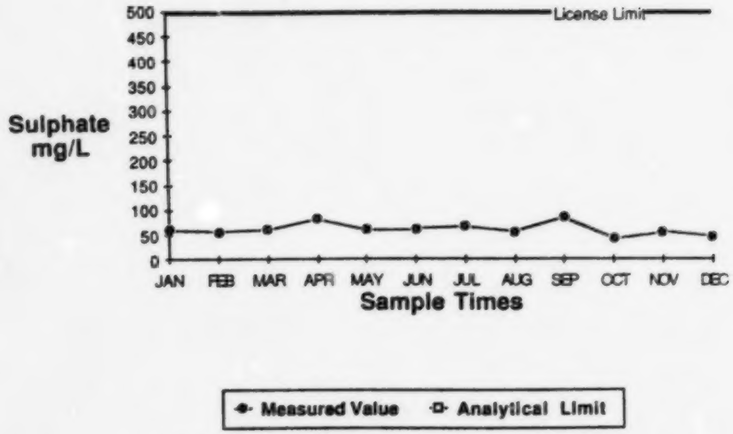


Fig. 8.33. **1985 - Treated Water- Rossdale**



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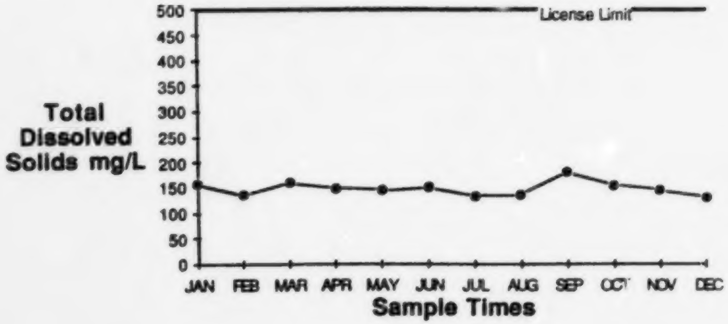
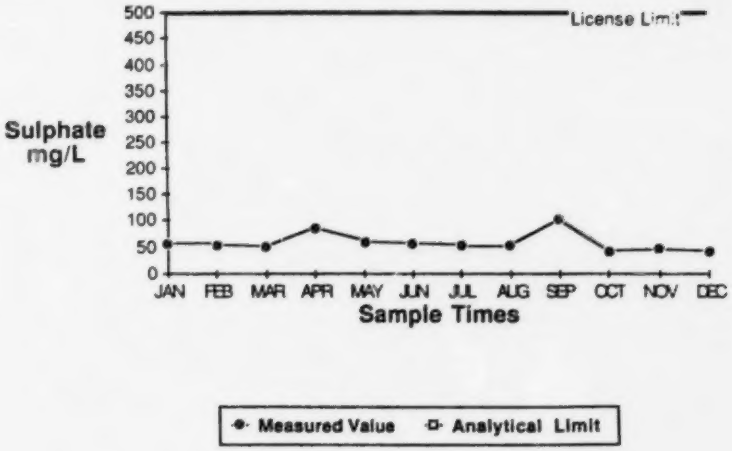


Fig. 9.33. **1985 - Treated Water- E.L. Smith**



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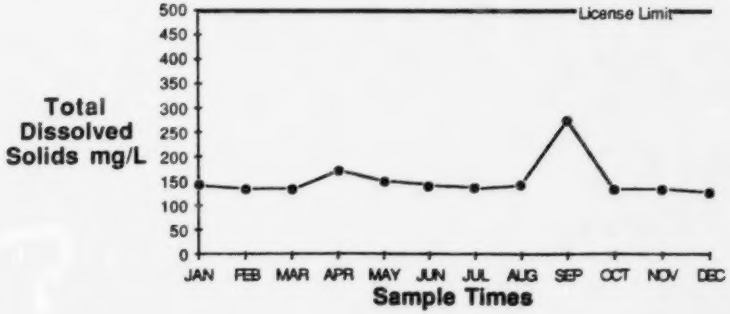


Fig. 8.35. 1985 Treated Water - Rossdale

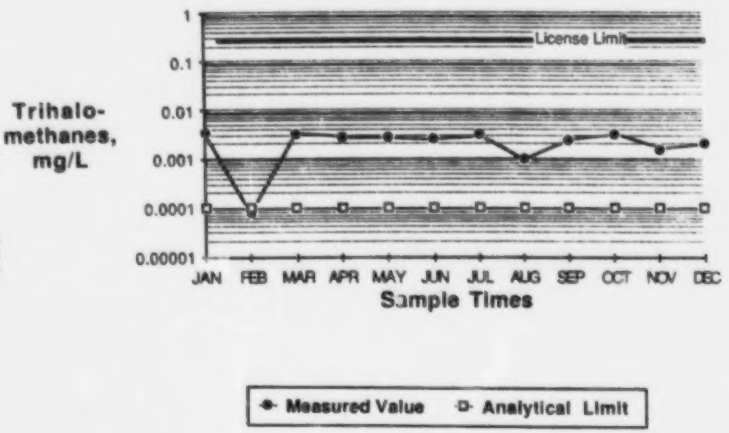


Fig. 8.36.

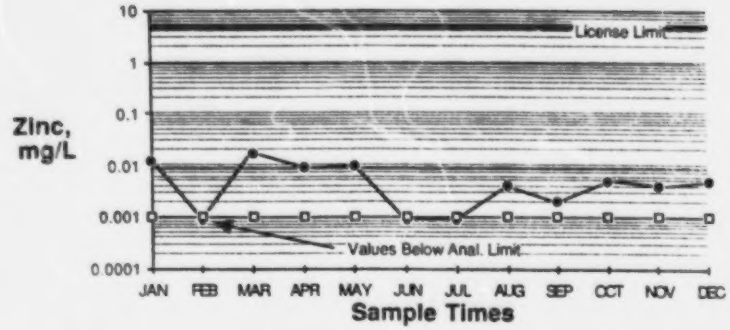


Fig. 9.35. 1985 Treated Water - E.L. Smith

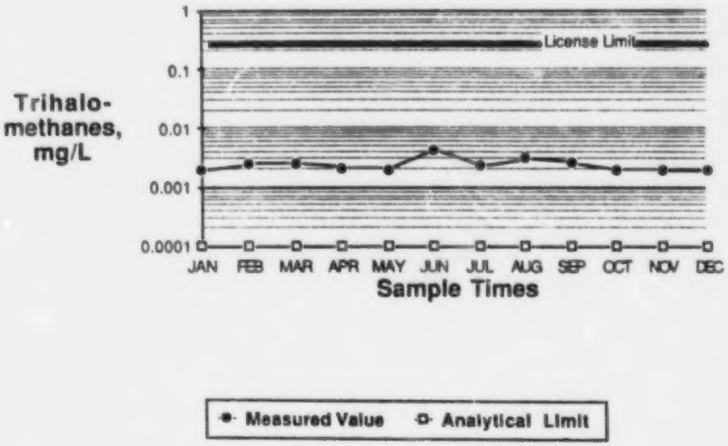
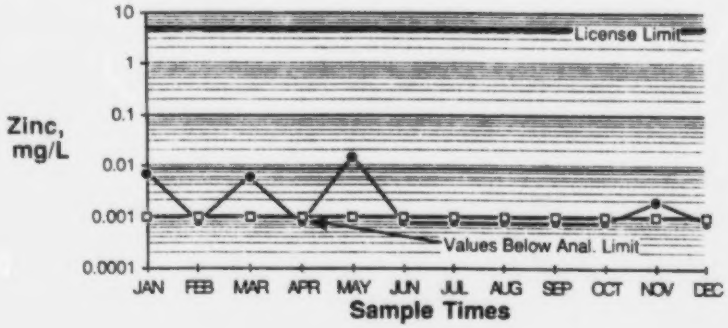


Fig. 9.36.



3.Samples are collected in the distribution system and consequently give information on the quality in the distribution system.

4.Analyses are done for many parameters which are not currently covered by guidelines.

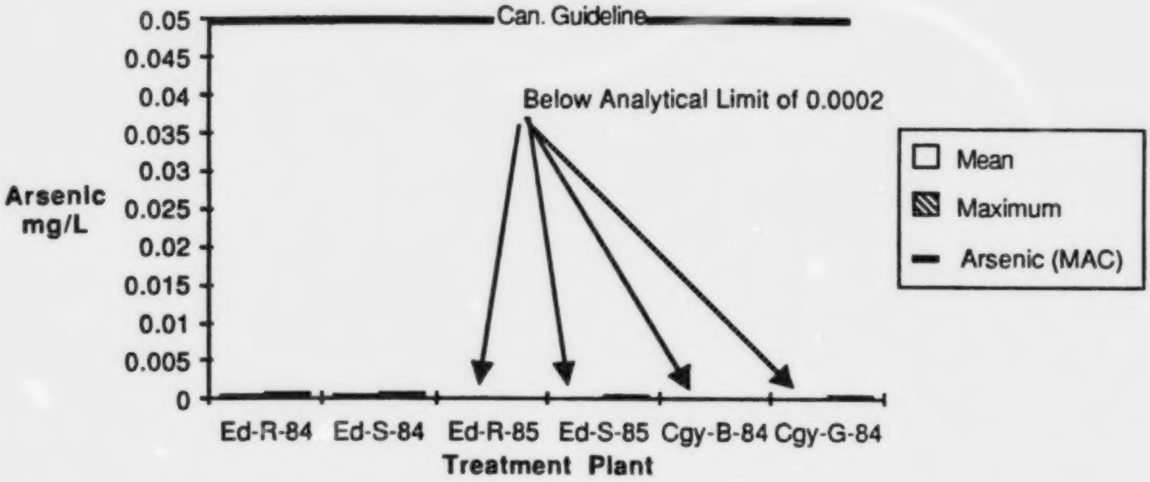
The results provided by the Alberta Treated Water Surveys[71-74] confirm the conclusions reached in section 3.2.2 that the quality of Edmonton drinking water for health-related license limits is excellent. With the exception of taste and odor, which is not part of the survey, results are also good for aesthetic parameters.

3.3 Edmonton Treated Water vs. Water from Elsewhere.

3.3.1 Edmonton vs. Calgary Treated Water Quality

Comparison of Edmonton and Calgary treated water was performed for 10 parameters which have health-related guidelines[10] and which were also available from the Alberta Treated Water surveys. Annual mean and maximum values are plotted for each parameter at both of Calgary's treatment plants (Glenmore and Bearspaw) and both Rossdale and Smith in Figures 10.1 to 10.5. The Calgary data and 1985 Edmonton data are plotted from Alberta Treated Water Survey data. The Edmonton 1984 data is taken from the 1984 Annual Report.[30] The latter is important in the cases of mercury and selenium where the City's data is higher than the Alberta Environment data (compare Figures 8.5 and 8.7 with Figures 10.2 and

Fig. 10.1. **Edmonton-Calgary Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = **Edmonton Rossdale, 1984**
Ed-S-84 = **Edmonton Smith, 1984**
Ed-R-84 = **Edmonton Rossdale, 1985**
Ed-S-84 = **Edmonton Smith, 1985**
Cgy-B-84 = **Calgary Bearspaw, 1984**
Cgy-G-84 = **Calgary Glenmore, 1984**

Fig. 10.2.

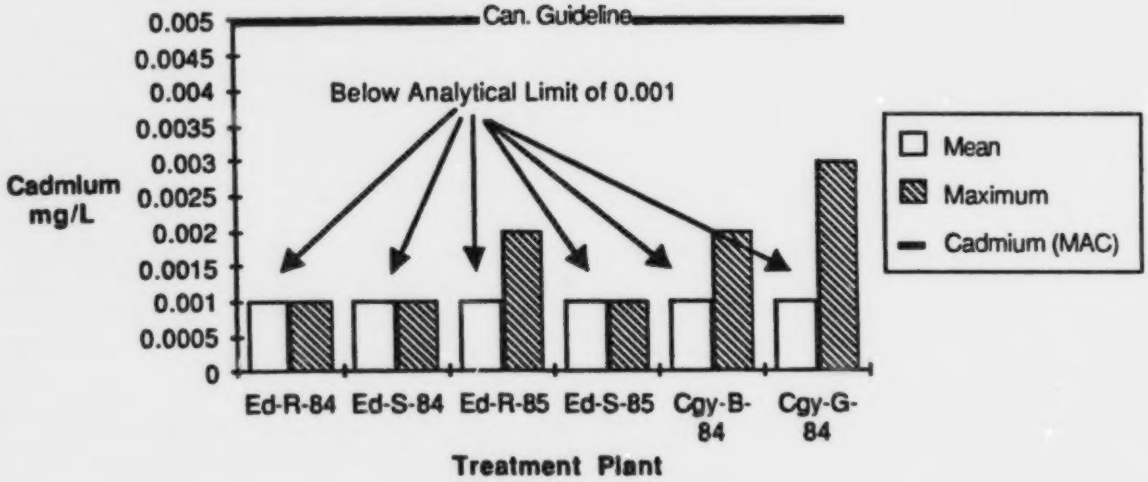
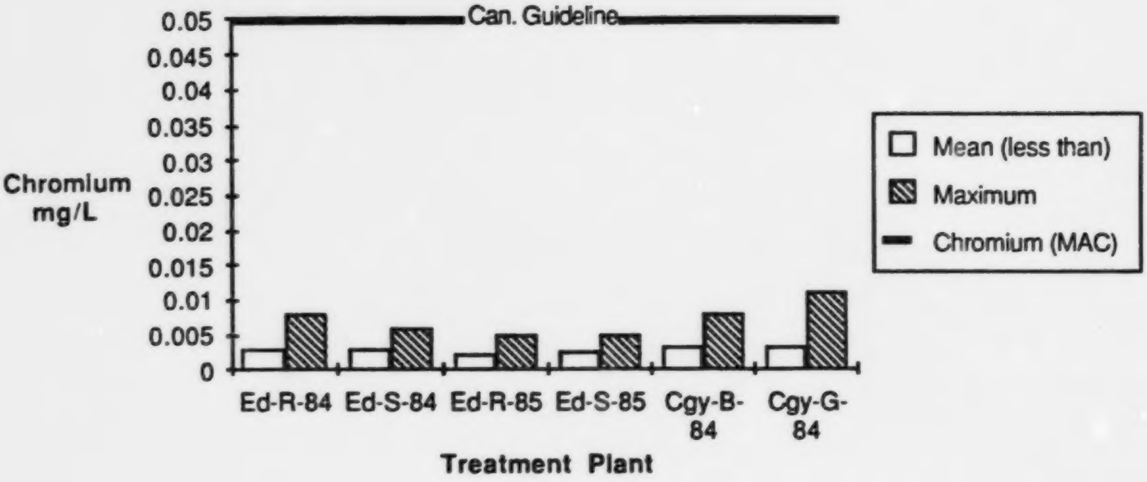


Fig. 10.3. **Edmonton-Calgary Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ed-R-84 = Edmonton Rossdale, 1985
Ed-S-84 = Edmonton Smith, 1985
Cgy-B-84 = Calgary Bearspaw, 1984
Cgy-G-84 = Calgary Glenmore, 1984

Fig. 10.4.

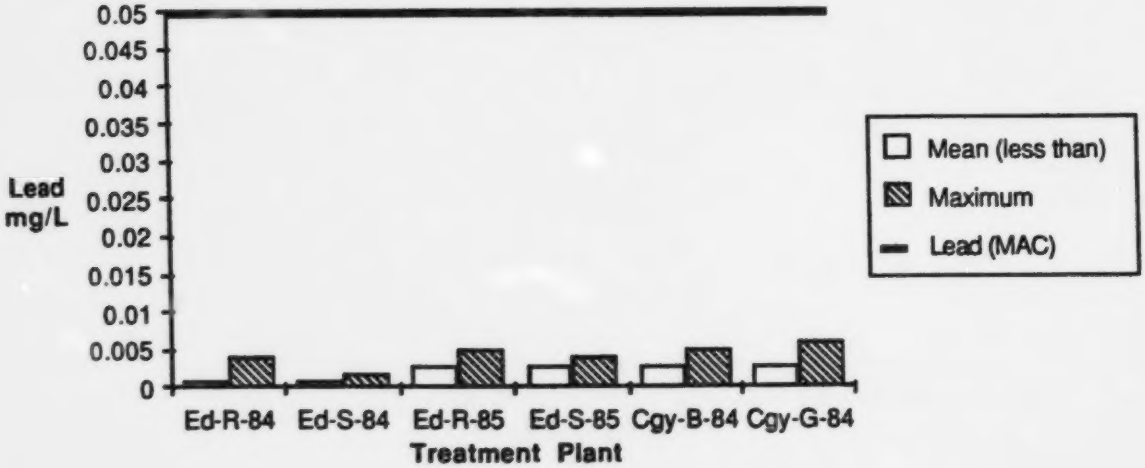
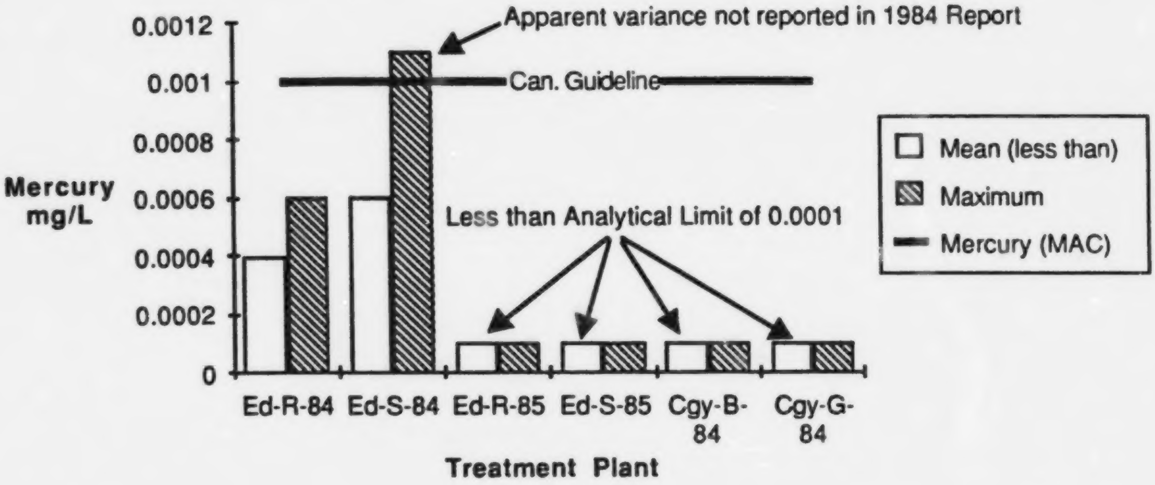


Fig. 10.5. **Edmonton-Calgary Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ed-R-84 = Edmonton Rossdale, 1985
Ed-S-84 = Edmonton Smith, 1985
Cgy-B-84 = Calgary Bearspaw, 1984
Cgy-G-84 = Calgary Glenmore, 1984

Fig. 10.6.

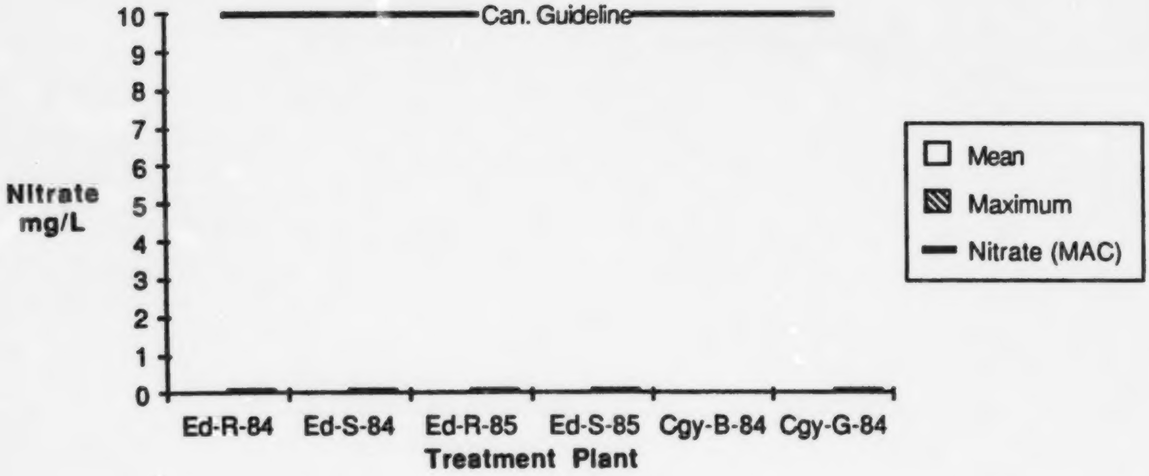
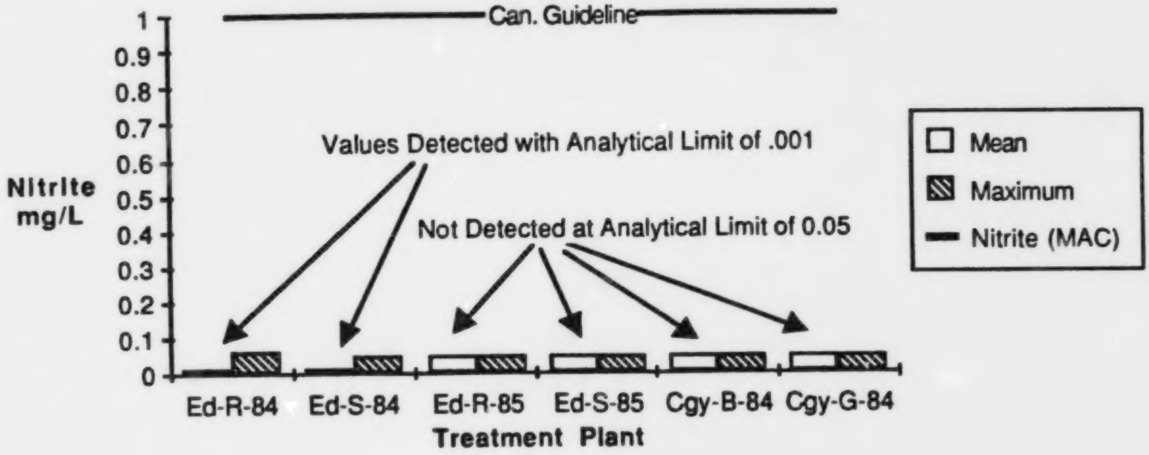


Fig. 10.7. **Edmonton-Calgary Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ed-R-84 = Edmonton Rossdale, 1985
Ed-S-84 = Edmonton Smith, 1985
Cgy-B-84 = Calgary Bearspaw, 1984
Cgy-G-84 = Calgary Glenmore, 1984

Fig. 10.8.

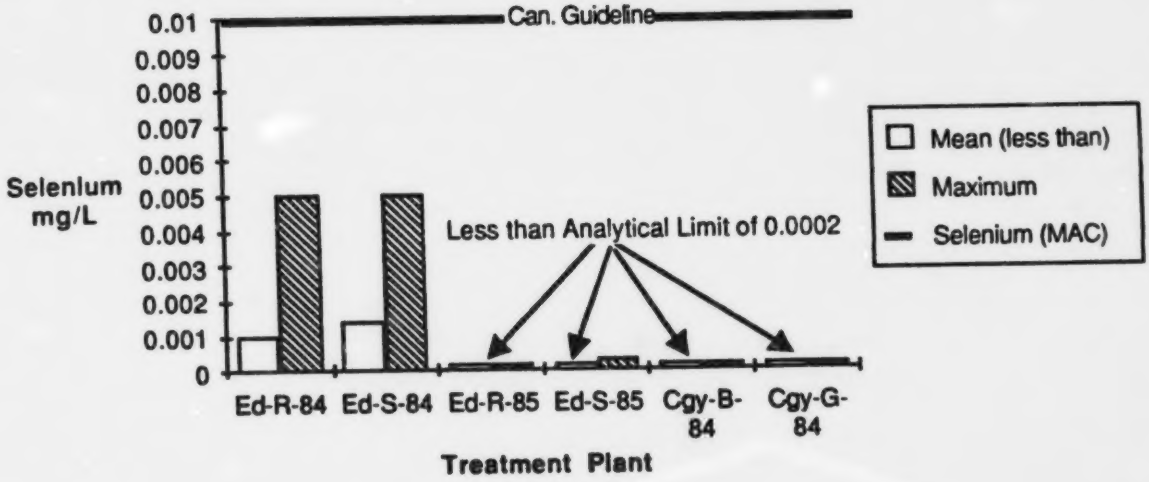
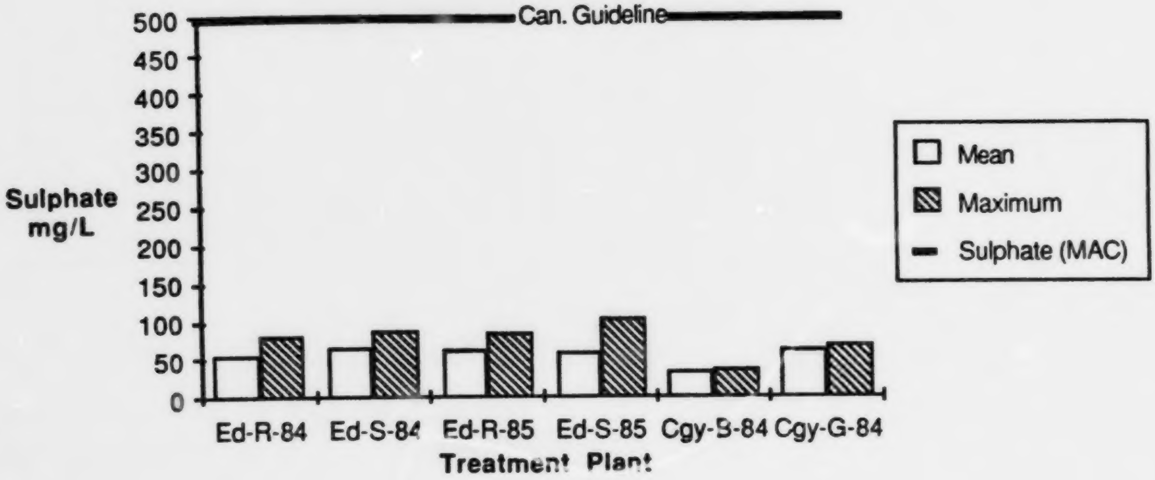
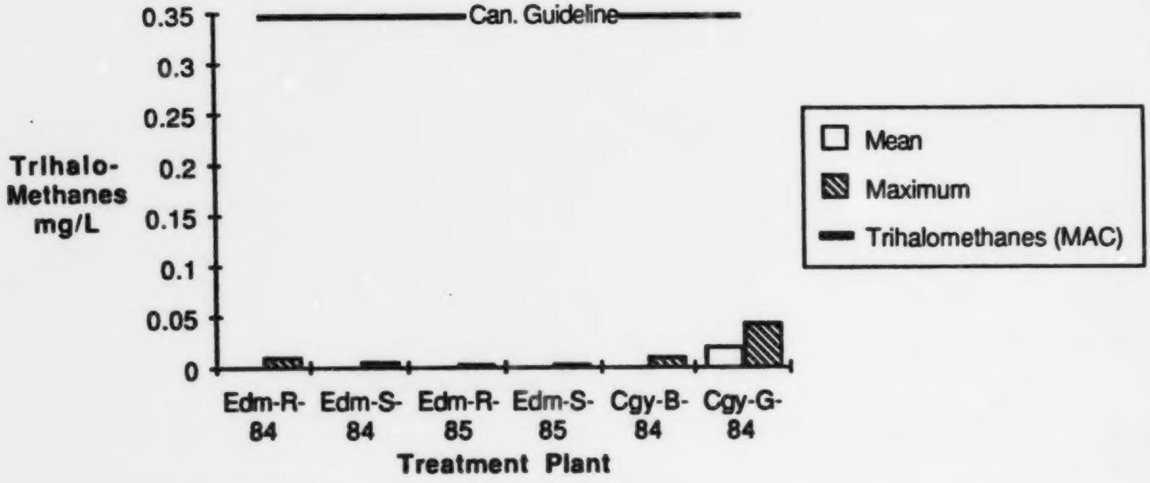


Fig. 10.9. **Edmonton-Calgary Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ed-R-84 = Edmonton Rossdale, 1985
Ed-S-84 = Edmonton Smith, 1985
Cgy-B-84 = Calgary Bearspaw, 1984
Cgy-G-84 = Calgary Glenmore, 1984

Fig. 10.10.



10.3). This has the effect of plotting a pessimistic scenario for Edmonton. Apart from the 1984 data for the high vlaues for those two metals, the comparisons do not show any superior quality for Calgary water.

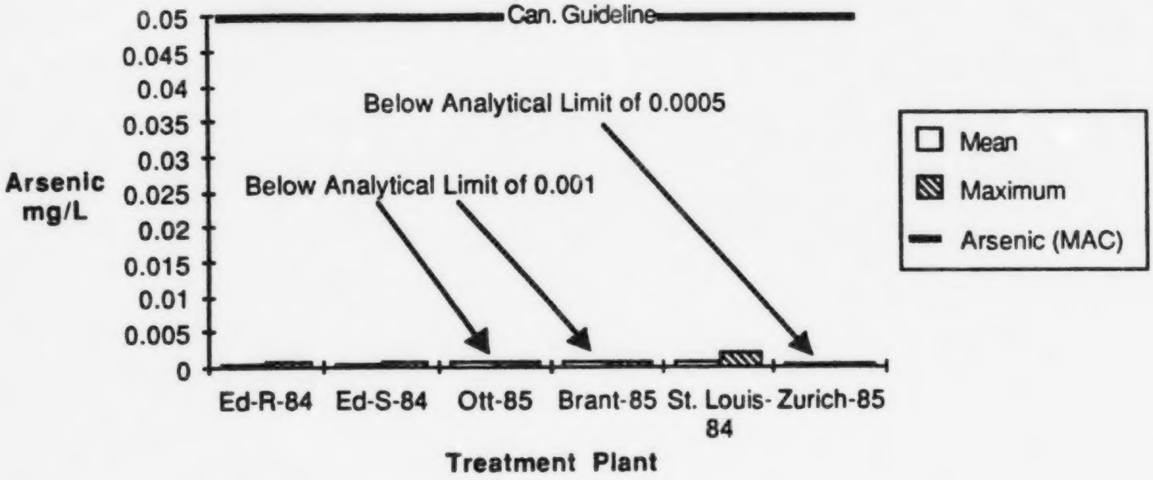
3.3.2 Edmonton vs Other Cities' Treated Water Quality

Edmonton was also compared with other cities which had been used as comparisons for treatment plants (see reports by Foley, Huck and Snoeyink, this Volume). For this comparison the data from the 1984 Edmonton Annual Report was used because the data from all other sources was produced by the municipality rather than the regulatory agency. These are plotted with annual mean and maximum values of 13 health-related parameters in Figures 11.1 to 11.7. These show that with the possible exception of silver, Edmonton's treated water is comparable with the comparison cities and in the case of nitrates Edmonton is noticeably better.

3.4 Trace Organics

A common criticism of the Candadian Drinking Water Guidelines is that they specify guideline levels for only a small number of organic contaminants. Because many of these have received increasing attention in recent years because of industrial accidents or hazardous waste incidents, the public is understandably concerned about the presence of trace organic substances in drinking water. This aspect of Edmonton's drinking water quality was considered in three

Fig. 11.1. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.2.

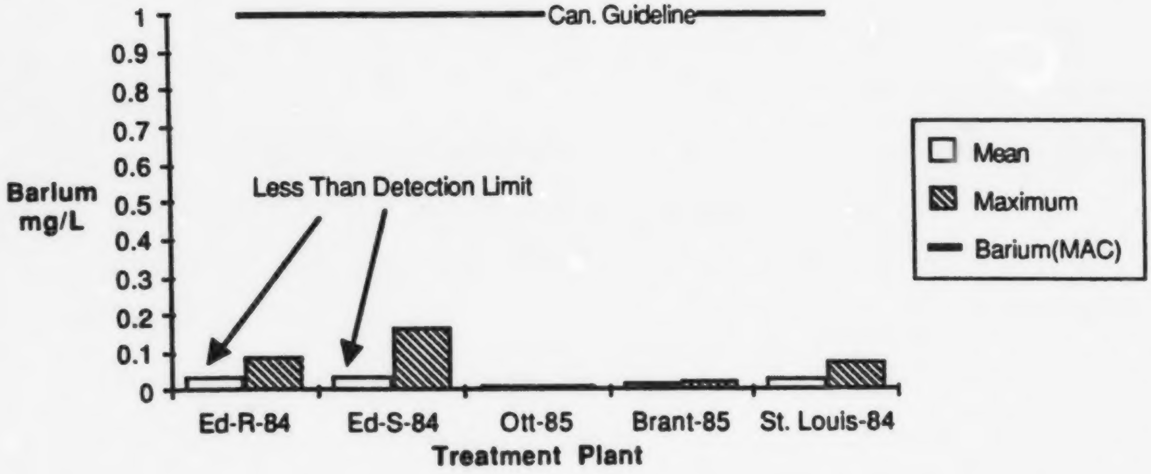
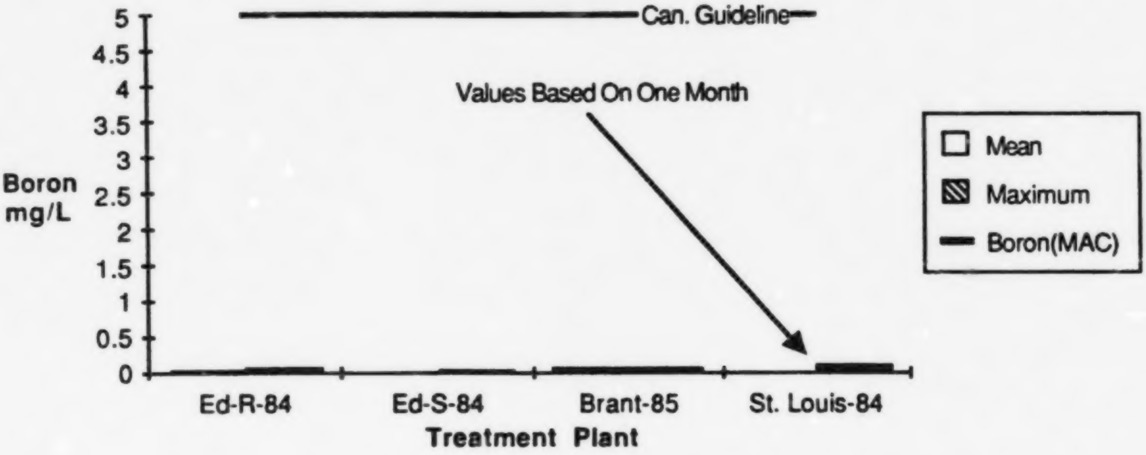


Fig. 11.3. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.4.

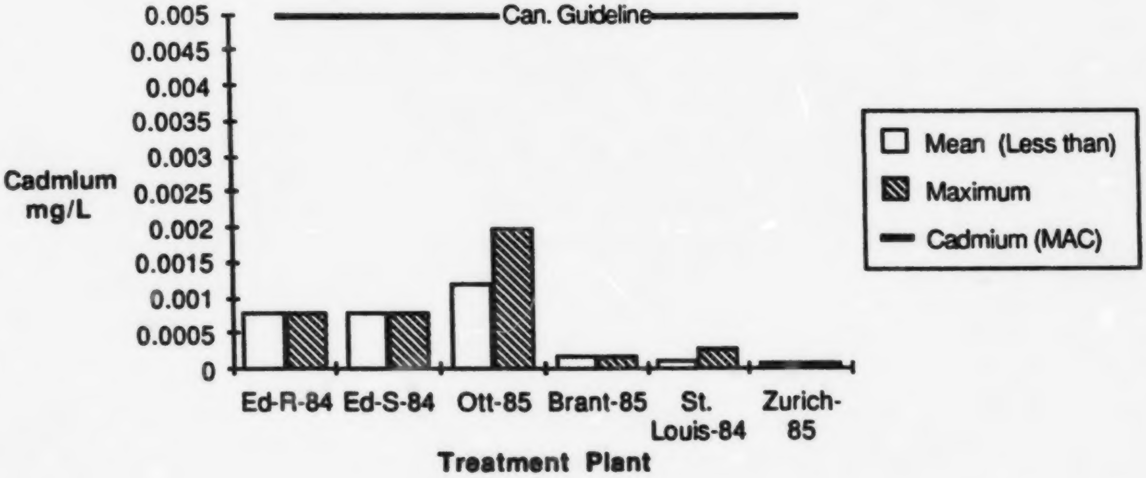
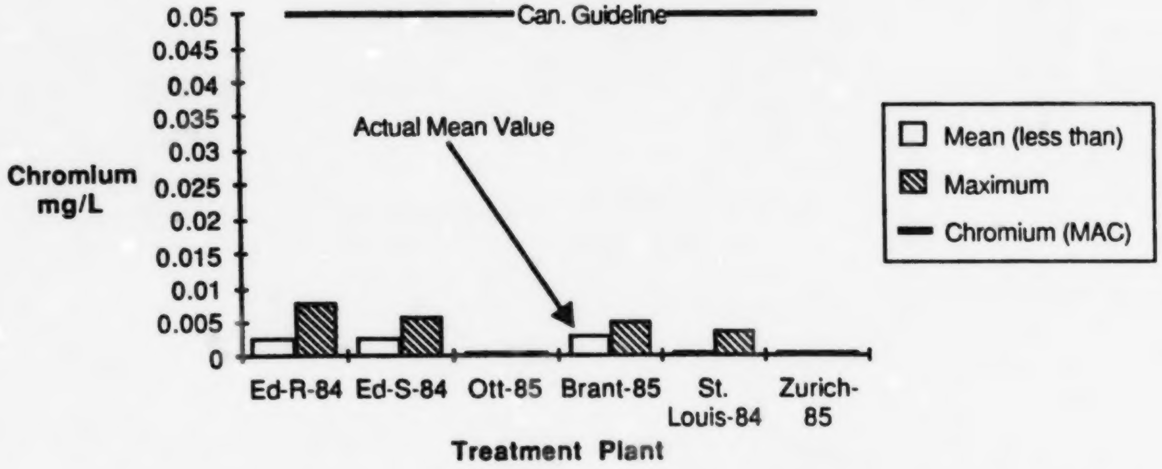


Fig. 11.6. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.5.

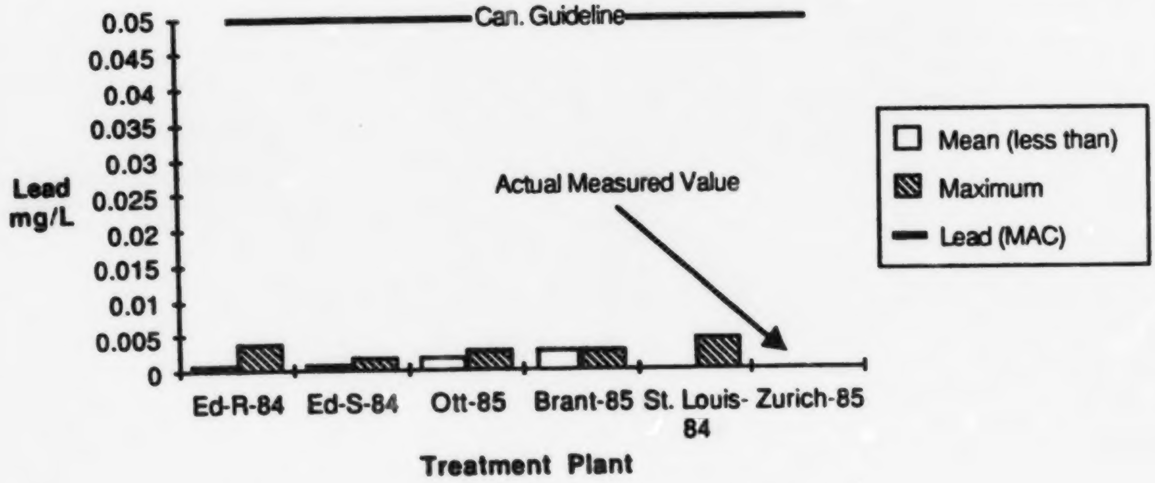
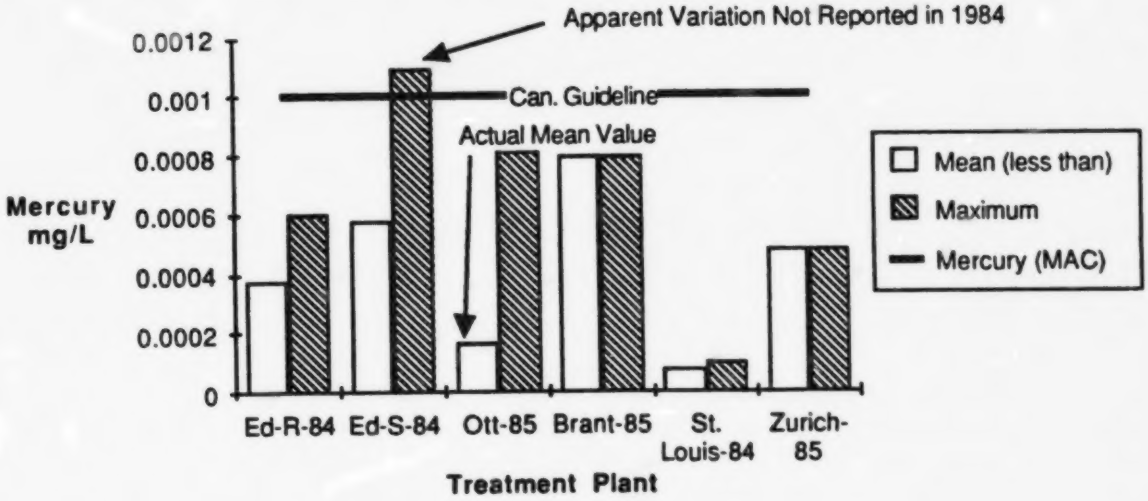


Fig. 11.7. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.8.

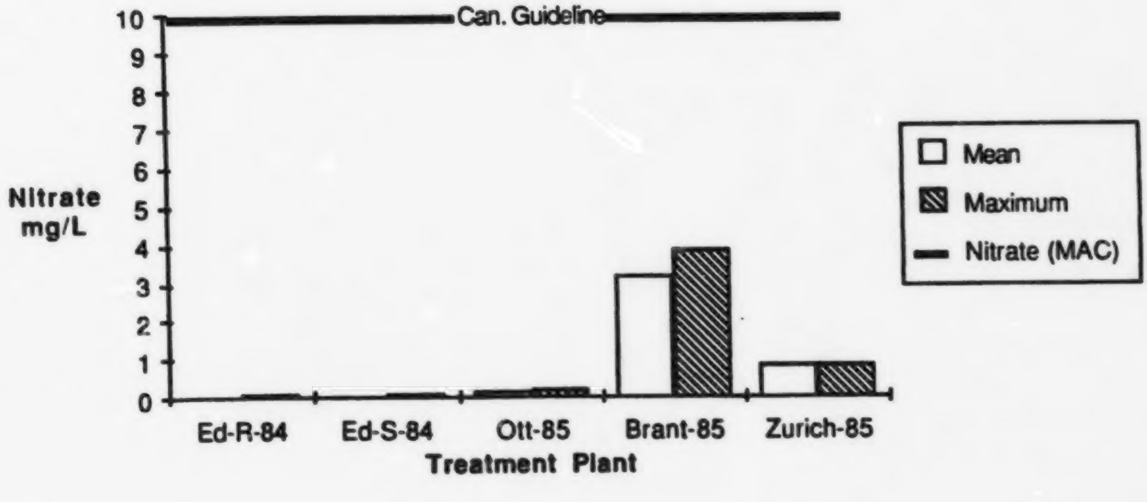
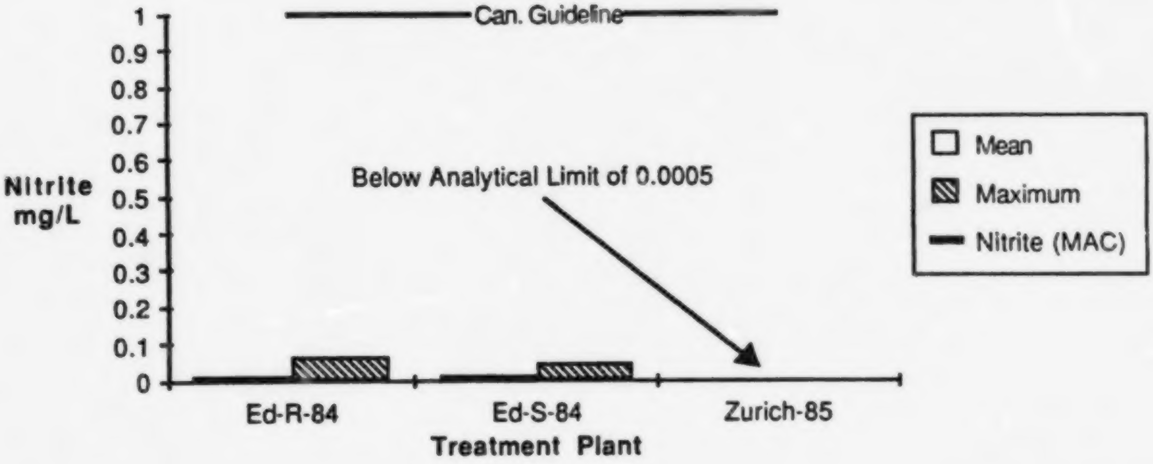


Fig. 11.9. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.10.

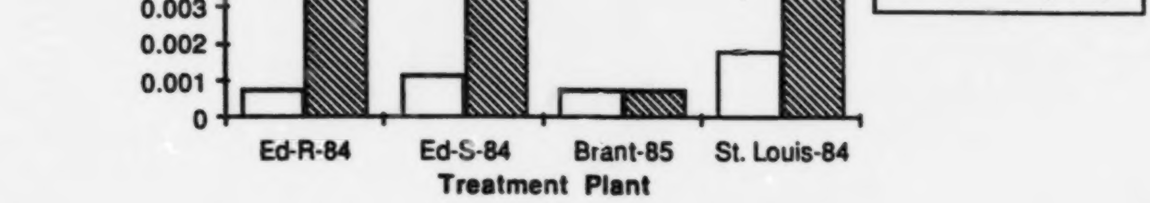
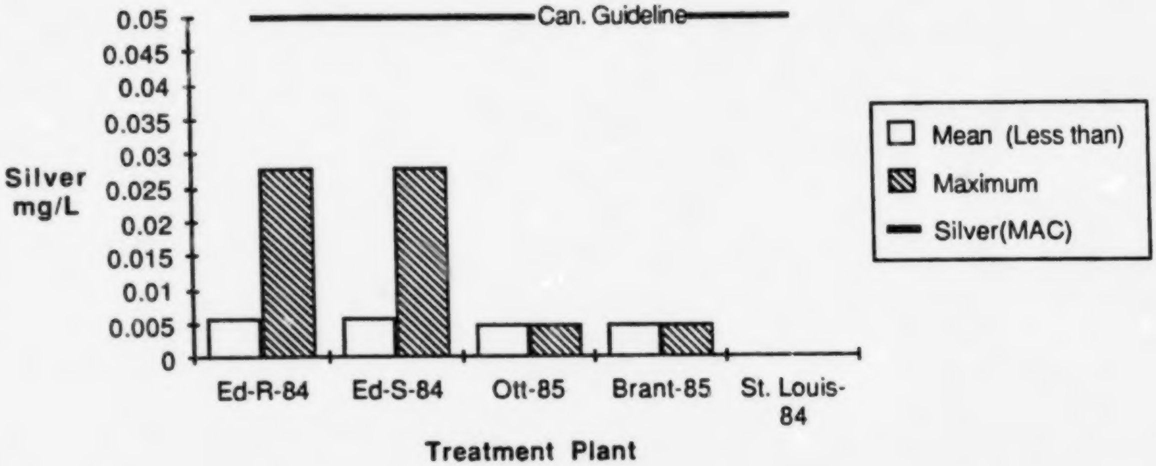


Fig. 11.11. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

Fig. 11.12.

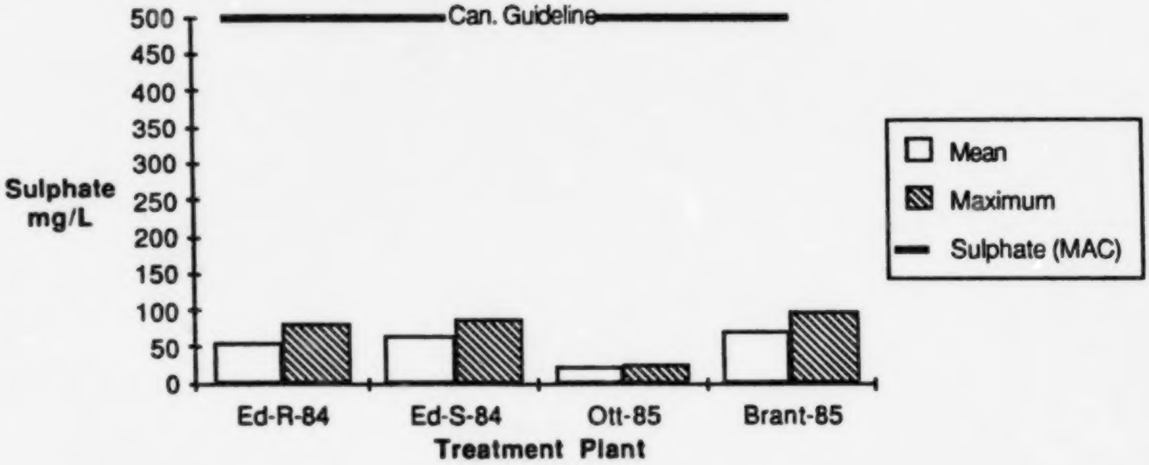
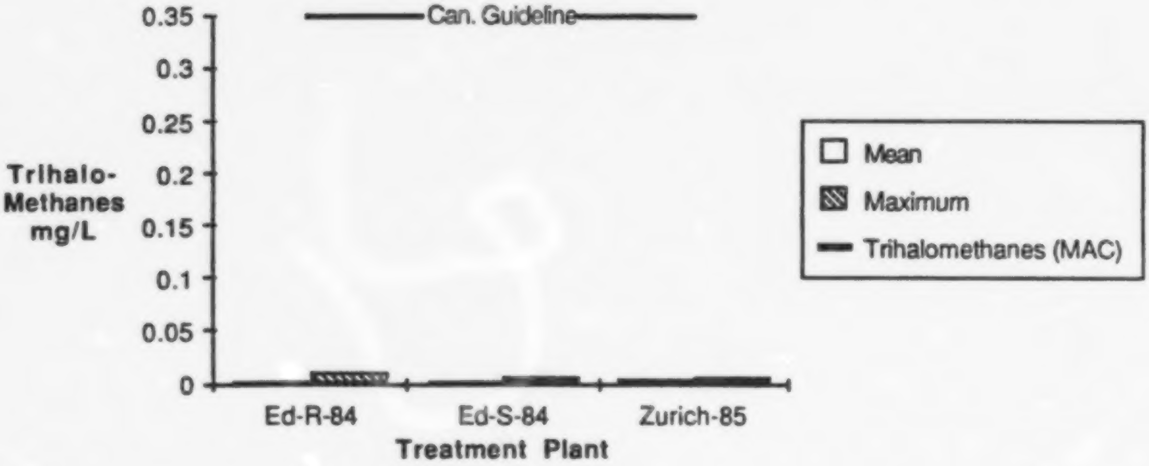


Fig. 11.13. **Edmonton Treated Water Comparison (Annual Mean and Maximum Values)**



Ed-R-84 = Edmonton Rossdale, 1984
Ed-S-84 = Edmonton Smith, 1984
Ott-85 = Ottawa, 1985
Brant-85 = Brantford, 1985
St. Louis-84 = St. Louis County, 1984
Zurich-85 = Zurich, Switzerland, 1985
Langenau-85 = Langenau, W. Germany, 1985

ways: review of earlier monitoring for trace organics in Edmonton water, review of Treated Water Survey results for Edmonton water and, discussion of 1986 monitoring survey results.

3.4.1 Previous Trace Organics Monitoring.

Apart from the Alberta Environment Treated Water Surveys, which will be dealt with in section 3.4.2, trace organics data on Edmonton water had been relatively limited. Edmonton has obtained at least as much information on trace organics as routinely obtained by Ottawa and Toronto, and likely more than most communities of its size.

The city undertook an initiative in the spring of 1982, submitting several samples for GC/MS analyses by Kinetic Environmental Research Laboratries.[35] As noted in section 2.6.1.1, these results must be regarded as tentative only because of the limited nature of interpretation done on mass spectra for compound identification and because of problems apparent with contamination of the solvent blanks. Many of the same compounds found in the raw water were also found in the treated water. Only the hydrocarbon identifications were generally correct according to Dr. Coutts' review of this data.

The City obtained analyses of 5 samples (raw and treated for Smith, raw, partially treated and treated for Rossdale) and submitted these to Envrio-Test laboratories for EPA priority pollutant analysis (base/neutrals and acid extractables, target pesticides and herbicides and non-target scan for base/neutral and acid fractions.[74] These analyses indicated no organics in any of the above categories for the Smith samples, either raw or treated and with the Rossdale raw sample. At Rossdale, the partially treated and treated samples exhibited hydrocarbons in the form of trimethylbenzenes at 10 ug/L(ppb) and 28 ug/L(ppb) but no organics in any other category.

A similar program in December 1984 considered Smith raw and treated, Rossdale raw and treated (plants 1&2 and plant3). These results[75] indicated the occurrence of ultra-trace levels (parts per trillion) of some chlorinated pesticides in treated but not in raw. All values were designated as being less than the method detection limit and not subject to confirmation by GC/MS results. These values are well below those specified by the Canadian Drinking Water Guidelines.[10] No organic compounds in the other categories were identified except for bis(ethylhexyl)phthalate and chloroform. However, the blank which accompanied these samples was contaminated with 150 ug/L(ppb) of organics which appeared in all samples and was subtracted out for the results reported. Also identified in the blank was 2,4,6 trichlorophenol.

Further demonstration of hydrocarbon impact occurred on February 28, 1985 when two treated water samples were analyzed by Enviro-test[76] using GC/MS. These were shown to contain 93 and 180 ug/L (ppb) of extractable hydrocarbons.

These results showed substantial quantities of alkylated benzenes and alkylated naphthalenes. The complete analyses are listed in Appendix F. These results caused considerable media and public concern when they were published in March.

The March monthly report[53] described the circumstances as: "Rising turbidity and color were observed in the raw water at E.L.Smith and Rossdale WTPs starting 02 27. This was the first urban runoff of 1985, bringing hydrocarbons into the river. The second runoff, from the watershed, started 03 19, causing on [sic] increase of turbidity and color. The elevated level of hydrocarbons resulted in complaints;" Clearly, treatment plant staff were able to recognize urban runoff as the source of the hydrocarbon contamination problem at Rossdale. Unfortunately, in 1985, hydrocarbons broke through into the treated water supply.

As noted above, on March 19, rural runoff hit and severe taste and odor problems arose at both plants. The GC/MS analyses[76] of extractable organics from Smith for March 21, 1985 showed four organic acids rather than hydrocarbons. These were: butyric acid, cyclohexanecarboxylic acid, benzoic acid and benzeneacetic acid (phenylacetic acid). At the same time, purge and trap GC/MS analyses for volatiles on a March 20, 1985 Smith sample identified four volatile compounds: 2-methyl propanal, chloroform, 3-methyl butanal and 2-methyl butanal. These aldehydes were identified with only limited confidence based only on library mass spectra matching at that time. Another sample taken March 1, at the Venture Inn hot water showed a mixture of these four compounds with a mixture of dimethyl and trimethylbenzenes. This was not received for analysis until March 28 and the analysis[77] was dated April 1. All the foregoing results are provided in Appendix F.

The health implications of the extractable compounds is dealt with in the reports by the Canadian Center for Toxicology and by Dr.Bull (this Volume). The aldehydes have been considered in only a preliminary manner at this time because their presence in Edmonton water was not confirmed until June 1986. Their significance to taste and odor is discussed in section 3.5.

Another small (with respect to Edmonton) scale survey was conducted in 1985 by the Department of National Health and Welfare.[78] Samples of raw and treated water were collected for Rossdale during the periods: October to December 1984, February to March 1985 and May to July 1985. These were analyzed for 34 halogenated phenols but detectable results occurred with only four at Rossdale: phenol, 2,4-dichlorophenol and pentachlorophenol. The first set showed none detectable, the second detected pentachlorophenol in the raw water at 0.000004 mg/L and the final set measured 0.000247 mg/L of phenol and 0.000071 mg/L of the dichlorophenol. Those levels are at or below normal detection limits used for plant monitoring. Hence, the findings are consistent, in a general way, with the routine monitoring data.

3.4.2 Treated Water Surveys

As noted in section 3.2 Alberta Environment has been conducting treated water surveys on larger Alberta communities since 1978. Results of the surveys[71-74] since 1983 were tabulated and are reproduced in Appendix E. These surveys represent a more consistent and extensive data base on trace organics than any I could acquire from any other jurisdictions.

Leaving the 1986 data for discussion in Section 3.4.3, a review of these summaries indicates very few identifiable organic compounds have been detected in Edmonton water, from either plant. These are summarized in Table 10. Those compounds with confirmed identification are generally present at low concentrations. These results indicate slightly fewer occurrences of trace organics at Smith compared with Rossdale.

The prevalence of identifiable organics in Edmonton water was no more common or significant than for the limited data acquired and reviewed from Ottawa and Toronto. The same can be said for a comparison with the Calgary plants, based upon review of the Alberta Treated Water Surveys. These generalizations cannot be extended to the problem samples obtained during spring runoff because analyses for problem circumstances at other cities was not available.

The health significance of the compounds which were identified is dealt with in the reports by the Canadian Center for Toxicology and by Dr. Bull (this Volume).

Table 10 Summary of Trace Organic Data - Alberta Treated Water Surveys

Compound	Rossdale 1983-85 36 samples Times Found	Rossdale 1983-85 36 samples Median Conc. ug/L	Rossdale 1983-85 36 samples Maximum Conc. ug/L	E.L.Smith 1984-85 24 samples Times Found	E.L.Smith 1984-85 24 samples Median Conc. ug/L	E.L.Smith 1984-85 24 samples Maximum Conc. ug/L
benzene	4	3.6	5.	2	0.2	0.3
bromodichloromethane	3	tr.	tr.	1	tr.	tr.
chlorobenzene	1	0.2	0.2	0	-	-
chloroform	35	2.9	55.	20	2	4.3
1,1 dichloroethane	1	0.8	0.8	0	-	-
ethylbenzene	3	1	1.5	1	0.3	0.3
methylene chloride	5	7.4	24.	2	1.	2.
tetrachloroethylene	4	0.2	0.3	0	-	-
toluene	2	0.6	1.	2	0.1	0.2
trichloroethylene	1	0.4	0.4	0	-	-
trichlorofluoromethane	4	0.4	0.8	1	tr.	tr.
o-xylene	3	3.1	12.	0	-	-
m-xylene	8	0.4	12.	2	0.7	0.9
p-xylene	3	1.5	12	3	tr.	0.5

tr.= trace, below reliable detection limit

Table 10 continued

Compound	Rossdale 1983-85 36 samples Times Found	Rossdale 1983-85 36 samples Median Conc. ug/L	Rossdale 1983-85 36 samples Maximum Conc. ug/L	E.L.Smith 1984-85 24 samples Times Found	E.L.Smith 1984-85 24 samples Median Conc. ug/L	E.L.Smith 1984-85 24 samples Maximum Conc. ug/L
2-chloronaphthalene	0	-	-	1	tr.	tr.
di-n-butylphthalate	3	tr.	tr.	4	tr.	tr.
dioctylphthalate	1	4.	4.	0	-	-
bis(ethylhexyl)phthalate	12	2.	7.8	11	2.5	6.

tr.= trace, below reliable detection limit

3.4.3 Monitoring Program - 1986

As described in section 2.5.3, the City of Edmonton undertook a massive sampling program during the 1986 spring runoff with an objective of better defining the nature of spring runoff and its impact upon raw water supply and treated water for the Rossdale and Smith plants. With regard to trace organics, gas chromatographic analyses were done for purgeable and extractable organics (320 analyses for the latter) and results for raw water were presented in Figures 6.1 to 6.3, respectively. These indicated the differences in raw water conditions between Smith and Rossdale and between the urban runoff period (declining by March 1) and the rural runoff period (rising on March 1).

Analysis for purgeable organics in treated water (Figures 12.1 and 12.2) showed consistently low levels near or below the 5 ug/L detection limit. This was true, even at Rossdale when the raw water showed values of up to 50 ug/L for purgeable organics. All the values appear low compared with semi-quantitative purge and trap gas chromatographic monitoring for volatiles by the City over the same time period. This likely reflects some losses by the carbon adsorption / solvent desorption method in comparison with the tenax / thermal desorption method used in the City lab. However, despite the possbility of low values, the trend of lower purgeable organics in treated samples than in raw samples seems clear.

Fig. 12.1. **Rossdale Raw Water and Treated Water: Purgeable Organics (February 25 to March 5, 1986)**

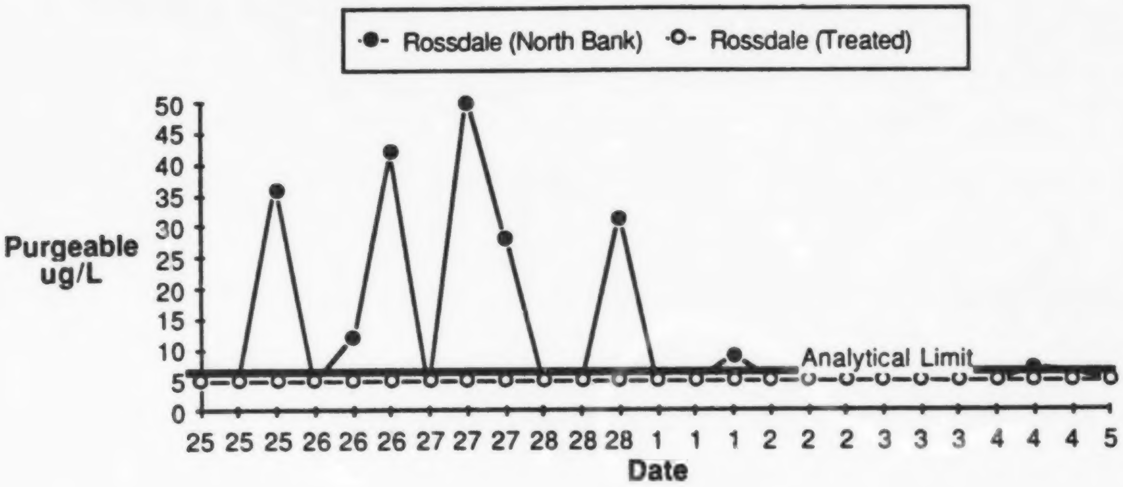
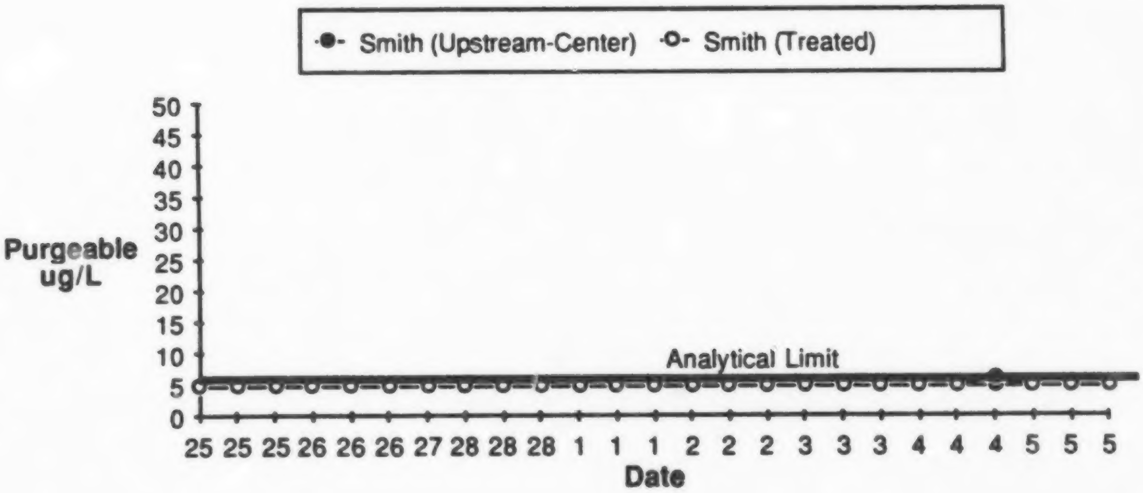


Fig. 12.2. **E.L. Smith Raw and Treated Water: Purgeable Organics (February 25 to March 5, 1986)**



The pattern is much clearer for the extractable organics, at least until March 2, because higher concentrations of extractable organics were found (Figures 13.1 and 13.2). At Rossdale, high quantities of extractables prevailed in the raw water up to March 1 without corresponding breakthrough into the treated water (Figure 13.1). This suggests that the powdered activated carbon addition was very effective in dealing with the raw water extractable organics, which were mainly hydrocarbons, before March 1.

During the corresponding period at Smith, there were very low levels of extractable organics in the raw water (Figure 13.2). After March 1, when the rural runoff hit, the pattern changed. Extractable organics rose in the raw water at both plants, and as noted in Table 9, the hydrocarbon character changed to predominantly organic acids in both raw and treated water. The latter are not effectively removed by either treatment plant. In fact, Figure 13.2 for E.L.Smith indicates that treated values for these compounds may be higher.

Although travel times through the plant were considered in staggering the sample times, some inaccuracy on those estimates could lead to anomalous comparisons of treated with raw concentrations. That concern seems minor for Smith, where the extractables in treated water more clearly exceed the raw, because the pattern for chloride progress through the plant appears reasonable (Figure 14.1). The higher chloride

Fig. 13.1. **Rossdale Raw and Treated Water: Extractable Organics (February 25 to March 12, 1986)**

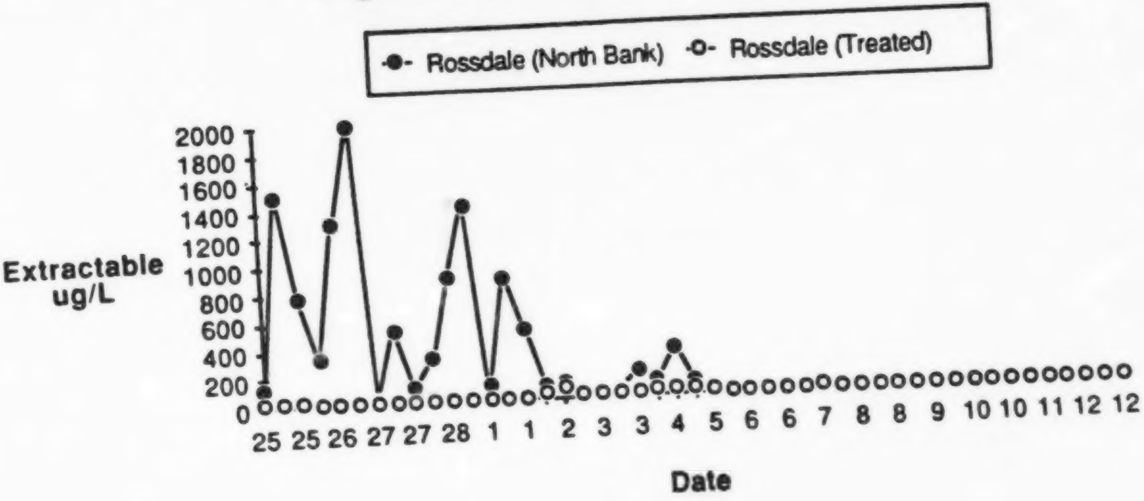


Fig. 13.2. **E.L. Smith Raw and Treated Water: Extractable Organics(February 25 to March 18, 1986)**

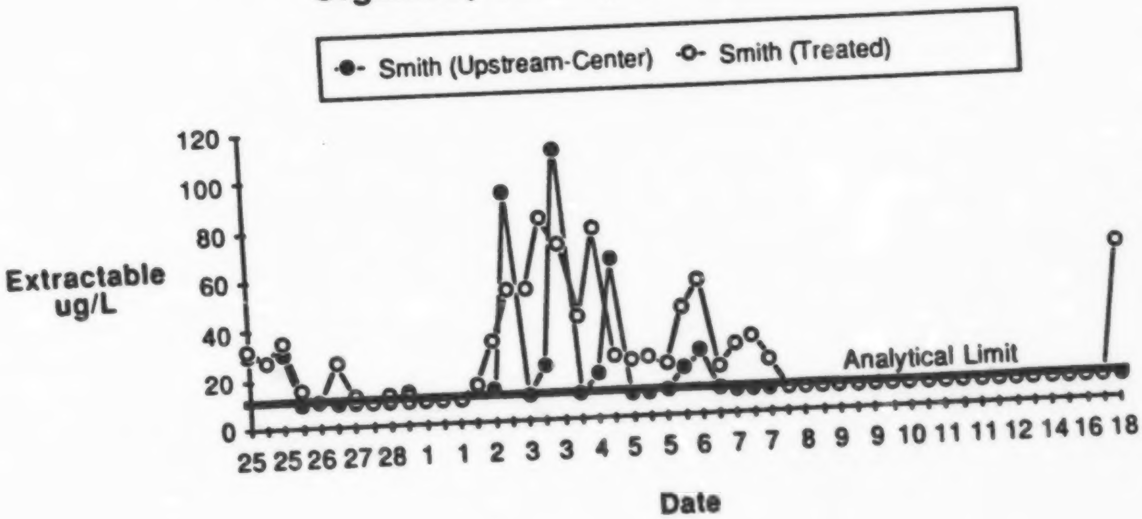


Fig. 14.1. **E.L. Smith Raw and Treated Water: Chloride (February 25 to March 5, 1986)**

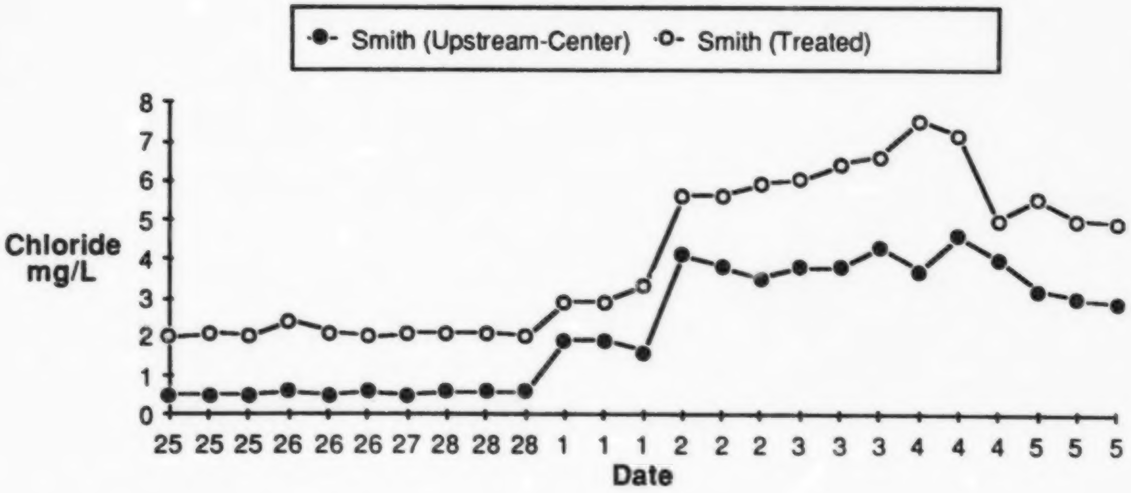
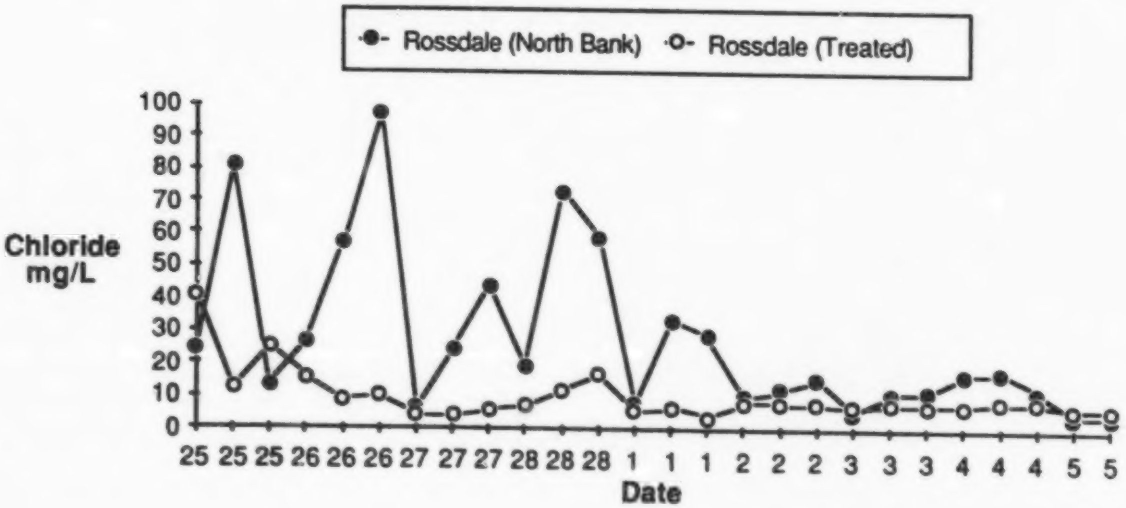


Fig. 14.2. **Rossdale Raw and Treated Water: Chloride (February 25 to March 5, 1986)**



values in treated than raw presumably reflect the addition of chloride arising from chlorine dioxide and excess chlorine end products. The correspondence of raw and treated patterns appears somewhat better than it may actually be, in that 16 hours pass between the last sample one day and the first sample the next. That lag period limits the tracking precision for step chloride increases, which did occasionally occur during the 16 hour period. The variable contribution of chloride from the process chemicals also interferes.

Similar correspondence at Rossdale is non-existent (Figure 14.2) which is an anomaly. There are no plausible sinks for chloride which can explain the apparent disappearance of chloride in the process. Because chloride is easily monitored, the City should follow up on this finding during next spring runoff to determine if there are some major anomalies in flow pattern in the plants.

The distribution of extractable organics among three sampling periods corresponding to peak urban, peak rural and post-rural runoff is summarized in Table 11. This summary suggests that higher values of extractables were present in the treated water than in the raw at least at Smith for the peak rural and post rural runoff periods. A paired t-test for the Smith raw and treated extractable organics indicates that treated values were significantly greater than raw ($p<0.05$).

This finding requires an explanation. Some extractables may arise directly from treatment chemicals such as coagulant aids. From March 1 to 6, activated silica was used but to no

Table 11 Extractable Organics Monitoring - Spring Runoff 1986

Plant - Source	Urban Peak (Feb.25-Mar.1) Samples #	Urban Peak (Feb.25-Mar.1) Mean ug/L	Urban Peak (Feb.25-Mar.1) Maximum ug/L	Rural Peak (Mar.2-6) Samples #	Rural Peak (Mar.2-6) Mean ug/L	Rural Peak (Mar.2-6) Maximum ug/L	Post-Rural (Mar.7-18) Samples #	Post-Rural (Mar.7-18) Mean ug/L	Post-Rural (Mar.7-18) Maximum ug/L
E.L. Smith - Raw	15	<14	30	15	29	110	23	<10	<10
E.L. Smith - Treated	13	<17	35	15	43	82	23	15	63
Rossdale - Raw	15	720	2000	15	74	340	26	129	32
Rossdale - Treated	15	<16	27	14	36	85	26	14	79

effect[65] so that it was replaced with the polymer, Percol on March 7. Reviewing Figure 13.1, the period of higher extractables in treated versus raw coincided with the use of activated silica. Intuitively, the organic polymer would seem to provide a more likely source of extractable organics than the silica. Without bulk chemical analysis on the silica, the possibity of extractables being present cannot be ruled out.

Another plausible explanation for higher extractable organics in the treated versus raw water would be the formation of organic acids from larger humic materials in the raw water by chlorine dioxide oxidation.

Overall, the extractable compounds identified in the treated water and listed in Table 9 are characteristic of oxidation products of natural organic matter. They can and, clearly by their identification in the raw water, do arise by natural oxidation processes. The extent to which they were increased by chlorine dioxide oxidation is not completely clear because of confounding factors. This could be clarified somewhat by carefully quantifying individual compounds in corresponding raw and treated samples. These compounds are unlikely to be contaminants in activated silica, so careful quantitative comparison could provide some insight.

Finally, a single sample of treated water collected from the distribution system on March 5, 1986 was subjected by Dr. Coutts at the University of Alberta, to different basic, acidic and acetylated extraction analysis procedures as described in Appendix G. These extracts were subjected to

GC/MS analysis and the results are reported in Appendix G. This approach confirmed the presence of many of the compounds reported in Table 9, but included many others, some which could not be identified and most of which cannot be pronounced. The results from the earlier monitoring are reviewed for their health significance in the reports by the Canadian Center for Toxicology and by Dr. Bull. Most of the additional compounds identified have no toxicological data available. This problem is addressed by Dr. Bull (this Volume)

3.5 Taste and Odour

3.5.1 Previous Experience

Taste and odour incidents in Edmonton's water supply have become engrained in local folklore as a rite of spring. Intensity of incidents has varied significantly from year to year, but some effect has usually been noticeable. Edmontonians may take heart from the reality that most water utilities experience taste and, or, odour problems at some time or another. An American Water Works Association survey of 120 utilities indicated that 87 experienced taste and odour problems at some time or another.[79] Even Calgarians occasionally experience odors from algal growth in the Glenmore reservoir.

However, the seemingly inevitable character of Edmonton's taste and odour problem has been frustrating. A detailed review of past events is difficult because relatively little rigorous or quantitative information has been gathered. However, a brief review is necessary to deal with our current and future problems.

Taste and odour problems in Edmonton were categorized into five major groupings: chlorophenolic, hydrocarbon, chloramine, metabolites-actinomycetes, and metabolites-algae.[80] These have been attributed to differing causes.

The chlorophenolic problem is associated with a medicinal taste which was blamed on the chlorination of natural or synthetic phenols in the water. In recent years, this type of problem has not been evident. The absence of a chlorophenol problem has been attributed to the use of chlorine dioxide which has been used in a major way at Edmonton plants since March 1982.[80] Unfortunately, there is no analytical data which directly relates the presence of specific chlorinated phenols in the water to taste and odour problems. A chlorophenol problem has been presumed primarily because compounds which respond to the standard colorimetric test for phenolics are occasionally present at concentrations above the Canadian Drinking Water Guideline[10] of 0.002 mg/L and chlorophenols are known to have detectable and characteristic medicinal taste at concentrations that low.[8] However, it is not certain that all substances responding to the standard colorimetric phenols analysis will necessarily form a foul tasting chlorophenol. Nor can the standard colorimetric test distinguish chlorinated from non-chlorinated phenols.

A direct determination of chlorinated phenolic compounds in water described as having a medicinal character could have provided substantive evidence of this problem but this type of data have not been collected. Such data is necessary to reliably implicate chlorophenols because non-chlorinated phenols cause detectable taste only at much higher concentrations than the levels which have been found in Edmonton water.

Hydrocarbons can cause taste and odour at extremely low levels. A few examples of threshold odour levels are:

diethyl benzene[31]	1.2 ug/L(ppb)

diesel[81]	0.5 ug/L(ppb)
ethyl benzene[31]	2.4 ug/L(ppb)
No.2 fuel oil[31]	0.8 ug/L(ppb)
naphtha[31]	16 ug/L(ppb)
naphthalene[81]	5 ug/L(ppb)
2-methylnaphthalene[82]	10 ug/L(ppb)
2,6-dimethyl-naphthalene[82]	10 ug/L(ppb)

These levels, when compared with some of the documented hydrocarbon intrusions of recent years, strongly suggest that hydrocarbons have been a cause of odour. Apparently, chlorine dioxide has not been effective in eliminating these compounds.[80]

Chloramines were used as the only disinfectant at Edmonton prior to the initiation of year-round use of chlorine dioxide in November, 1983. However, chloramines are still maintained at a target of 2 mg/L, total residual, leaving the plant. Chloramines used at higher levels can produce a strong chlorinous odour. Occasionally during problem circumstances, such as those plaguing plant 3 in 1985, higher doses were necessary to assure meeting the bacteriological standards. Such incidents can produce taste and odour complaints.

During runoff events, the river carries drainage from decomposing organic matter which may have collected on land. These materials can cause musty or earthy odors in the water (see Brownlee report, this Volume). Such problems are common to any surface water supply, but they are usually transient in a flowing supply.

Finally, because the coagulation and softening treatment tanks at the Rossdale plants 1&2 are exposed to sunlight, they have experienced severe algae growth in the past. Since chlorine dioxide has been applied to the raw water, this growth has been eliminated. Algae growth can give rise to a variety of taste and odour problems but those mentioned for Edmonton were described as a fishy taste and odour.[80]

Of these problems, the hydrocarbon related events had been the most severe in the past few years. Such problems were largely restricted to Rossdale because of its location downstream from the 85 storm sewer outfalls. However, around March 20, 1985, intense odour problems struck, coincident with the arrival of upstream runoff (as indicated by extremely high color in the raw water). The operational mode of dosing chlorine dioxide as necessary to maintain a residual of 0.1 mg/L resulted in doses as high as 1.8 mg/L at Smith and 2.2 mg/L at Rossdale plant 3. This event was somewhat unique in that, previously, the Smith plant had been somewhat immune to severe taste and odour events. Those which had occurred were limited to relatively rare and slight musty odour complaints. The cause for the March 1985 problem did not fit neatly in any of the previous categories.

Media coverage at the time of the events was preoccupied with the analyses from Rossdale in late February which had shown high levels of hydrocarbons.[76] Prior concerns with phenol-related taste and odour led to considerable discussion of phenols. However, the actual odour was different from those types of incident. Descriptions included: musty, woody, bleachy and swampy. The events passed without any clear indication of the specific offending compounds.

3.5.2 Taste and Odour in March 1986

The events following March 1, 1986 appear very similar, in retrospect, to the taste and odour experience of March 1985.

The major problem arose at a time of rapidly rising raw water color, dissolved organic carbon and corresponding chlorine dioxide demand. In March 1986, chlorine dioxide doses reached a maximum of 2.75 mg/L before the new Alberta Environment specification of a maximum of 0.5 mg/L residual chlorine dioxide plus chlorite was implemented on March 5.

The progress of the odour problem was clearly demonstrated by monitoring threshold odour number (TON). This represents a ratio:[12]

> [volumes of sample plus odour free dilution water
> necessary to just achieve a detectable odour]

divided by:

> [original sample volume].

For a sample with no detectable odour, it would be noted as such. A sample which is just detectable at full strength but which becomes non-detectable with any dilution would represent a threshold odour number of 1. If dilution with an equal volume of odour free water is just detectable, the TON is 2 and so on. This type of quantitative assessment is very time-consuming and has not been performed in recent years. However, it is extremely useful for tracking the course of an odour incident.

Figure 15.1 and 15.2 present threshold odour numbers for the E.L. Smith and Rossdale plants from February 25 to March 8. Samples of treated water were collected three times a day to coincide with the other sampling program. Unfortunately resources did not allow performance of TON on raw water thoughout, but once the odour event became evident, sampling of raw water commenced on March 3 at Rossdale and March 4 at Smith. These data clearly show how quickly and how intense the odour incident was. The peak of odour intensity corresponds closely with the peaking of dissolved organic carbon (Figure 6.5) and the UV absorbance (Figure 6.6) measures.

These data also confirm that there was little problem with odour during the first week when urban runoff was affecting the Rossdale intakes but not the Smith intake. These data, along with the earlier data on purgeable and extractable organics suggest that powdered activated carbon treatment was very effective at Rossdale in 1986. However, when the rural runoff hit, the odour problem became equally intense at both plants.

The data are less certain, because of the limited data set, about the relative intensity of raw water versus treated

Fig. 15.1. **Threshold Odour Number - E.L. Smith**

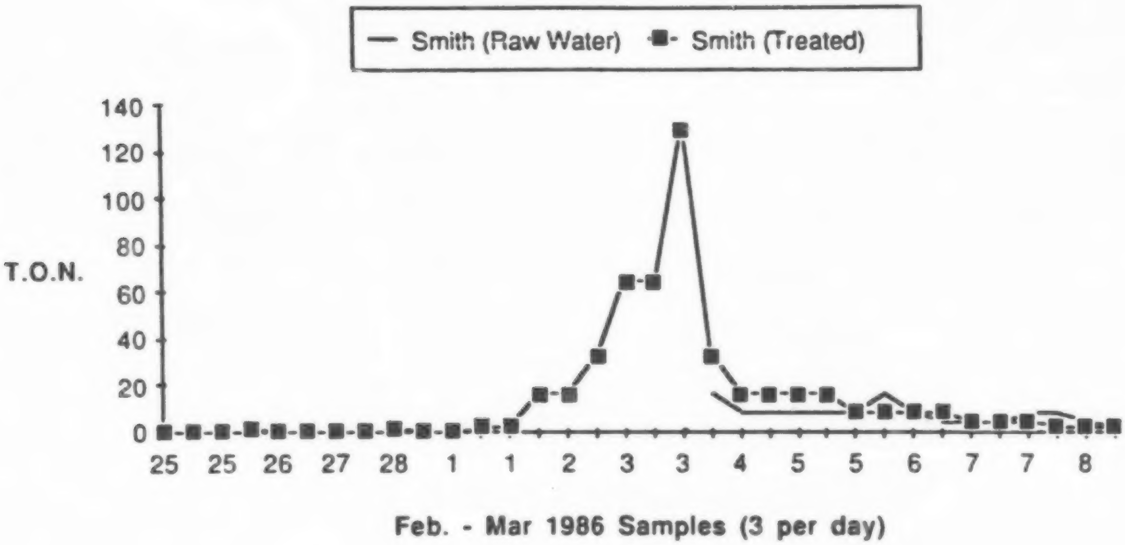
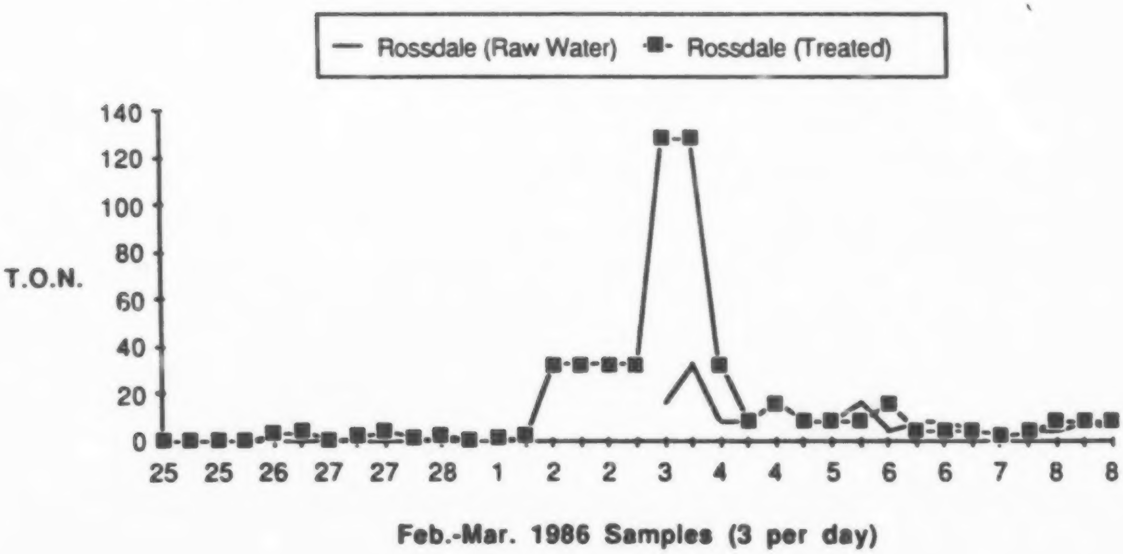


Fig. 15.2. **Threshold Odour Number - Rossdale**



water odour. However, the March 3 and 4 data seem to indicate a more intense odour in the treated water. Definitely, the odors were qualitatively different with the raw water generally assessed as musty. The treated water was much more complex in its character.

An assessment of the cause of the musty odour was attempted by sending raw and treated water samples from March 7 and 8 and a 16 L sample from March 4, to the National Water Research Insititute in Burlington. These were subjected to analysis for the trace metabolites geosmin and 2-methylisoborneol (MIB) which are recognized for their musty odors at extremely low concentrations. The findings are summarized in Dr. Brownlee's report (this Volume). He found trace levels of geosmin just above the detection limit of 0.01 ug/L (about the odour threshold level) for all raw and treated samples from March 7. Geosmin was only detected in the treated water from Rossdale on March 8. Dr. Brownlee was unable to obtain a clean mass spectrum to confirm identity of MIB but appropriate fragments were present and GC retention times for authentic standards matched the suspect peak for three different columns. The concentrated extract from the 16L sample has been returned to the University of Alberta and is being subjected to further evaluation.

With regard to the treated water, a panel of assessors from the University of Alberta Department of Foods and Nutrition[83] described the odour with a wide range of terms including: chlorine, earthy, stale, disinfectant, bitter, ammonia, organic, muddy, moldy and bleach-like. Flavour descriptors were: stale, earthy, organic, muddy, metallic, chemical, marshy, moldy, musty, sloughy, sewagey, decayed plant-like and oily.

Clearly, the problem was one which was perceived very differently by different people, even when exposed to exactly the same water. This finding highlights the difficulty associated with attempting to specifically identify problem agents strictly on the basis of sensory perception.

Although these events occurred during the intensive City monitoring program, the compounds which could be identified in the extractable organic samples could not explain the type of odour perceived. The purgeable organics analysis which should have shown peaks corresponding to the problem compounds were relatively clean. Only chromatograms run with the City Water Lab's volatile organic analysis scheme showed the presence of volatile organics coincidin with water complaints. Unfortunately, this method has no means for providing confirmed identifcation of peaks.

At the suggestion of A.R. Trussell, our expert on taste and odour (see Trussell report, this Volume) the City water labs acquired standards for three aldehydes which had appeared in trace quantity in 1985 samples.[76] As well, a sample of treated water sampled from the E.L Smith plant at a tap in Riverbend (March 3, 1986) was submitted to Enviro-Test for purge and trap analysis linked to GC/MS for compound identification. The results, provided in Appendix H, confirmed substantial quantities of the three short chain aldehydes: 2-methyl propanal (isobutyraldehyde), 2-methyl butanal and 3-methyl butanal (isovaleraldehyde). The actual quantities reported cannot be relied upon for an assessment of actual levels experienced in March because analyses were not performed on this sample until June 10, 1986. Although the sample was refrigerated in the dark, it likely contained some chlorite and possibly some chlorine dioxide residual. These could have reacted to change sample composition over the 3 month storage period.

The sensitivity of samples to storage is evident from a summary of analyses performed on similar samples from March 3 and March 5, 1986 by Montgomery Laboratories, Pasadena, California (Appendix I). These samples were in transit without refrigeration, for 2 days. They confirm the presence of traces of 3 and 2-methyl butanal but no 2-methyl propanal. On the other hand they show the presence of substantial quantities of methylethyl ketone which was not present in the samples kept under refrigeration until analysis. A raw water analysis failed to indicate the presence of any of the aldehydes, but this sample would be even more unstable because of the assured presence of bacteria. Finally, this sample suffered from contamination with acetone, presumably in preparation, shipping or analysis.

A review of the record of volatile organic analyses obtained by the Edmonton water labs, shows the presence of unidentified peaks in treated water samples, at about the right retention times, to correspond with the aldehydes. This provides only presumptive evidence of their presence at the time of events. However, the pure compounds have been prepared in a cocktail and assessed against individual recollection of the odour recalled for March. Most people exposed informally to the cocktail recognize it as the odour they recalled. In some cases, recognition was immediate and very definite (i.e. "that's it!!!"). Unfortunately, because the compounds were not expected as the problem source and there was no facility available for extensive purge and trap GC/MS during the monitoring survey, we do not have direct confirmation of the degree to which these compounds caused odour problems in 1986. The rapid peak and decline of the TON (Figure 14.1 and 14.2) suggests that samples had to have been collected at the right time. However, there are several additional factors which strongly suggest these compounds were a significant factor in causing odour problems for many Edmontonians in March 1986 and likely in March 1985.

As noted in section 3.5.1, these compounds were confirmed as present in trace quantities, together with traces of purgeable hydrocarbons, in an a 4 week old sample from March 1, 1985. They were also found without quantitation (because standards for these compunds were not available at the time) in a fresh sample on March 20, 1985. Experience in 1986, when standards have been available to deduce concentration, indicates that routine, broad spectrum analysis for these compounds is not likely to be very sensitive. Estimates of their quantity based upon comparison with hydrocarbon standard response will likely suggest lower concentrations than actually exist. They may even be missed if routine GC/MS scans are started at m/e above 45 because the main fragments occur below that number (D. Smillie, personal communication, D. Erickson, personal communication).

These compounds have been reported as produced by chlorine dioxide oxidation of Ohio River water.[84] This work, reproduced in Figure 16, shows the enhancement of several aliphatic aldehydes in response to chlorine dioxide treatment, but the same materials were already present in the raw water to some extent. Poor response of the aldehydes relative to other compounds present in these samples indicated the need to monitor specifically for the m/e 29 fragment which produced Figure 16. Without this enhancement it was difficult to notice the aldehydes against the background of other organics. Because of the preliminary nature of these studies, the authors cautioned against using the quantitative comparisons for too much interpretation. However, they supported the observation that aldehydes were increased from the raw water in their case. Stevens et al.[84] propose that the aldehydes are likely formed from olefins or amines (with reactivity being tertiary>secondary>primary).

The presence of these aldehydes in Edmonton water was not significant of itself without some indication that they are capable of causing odour at low levels. That confirmation

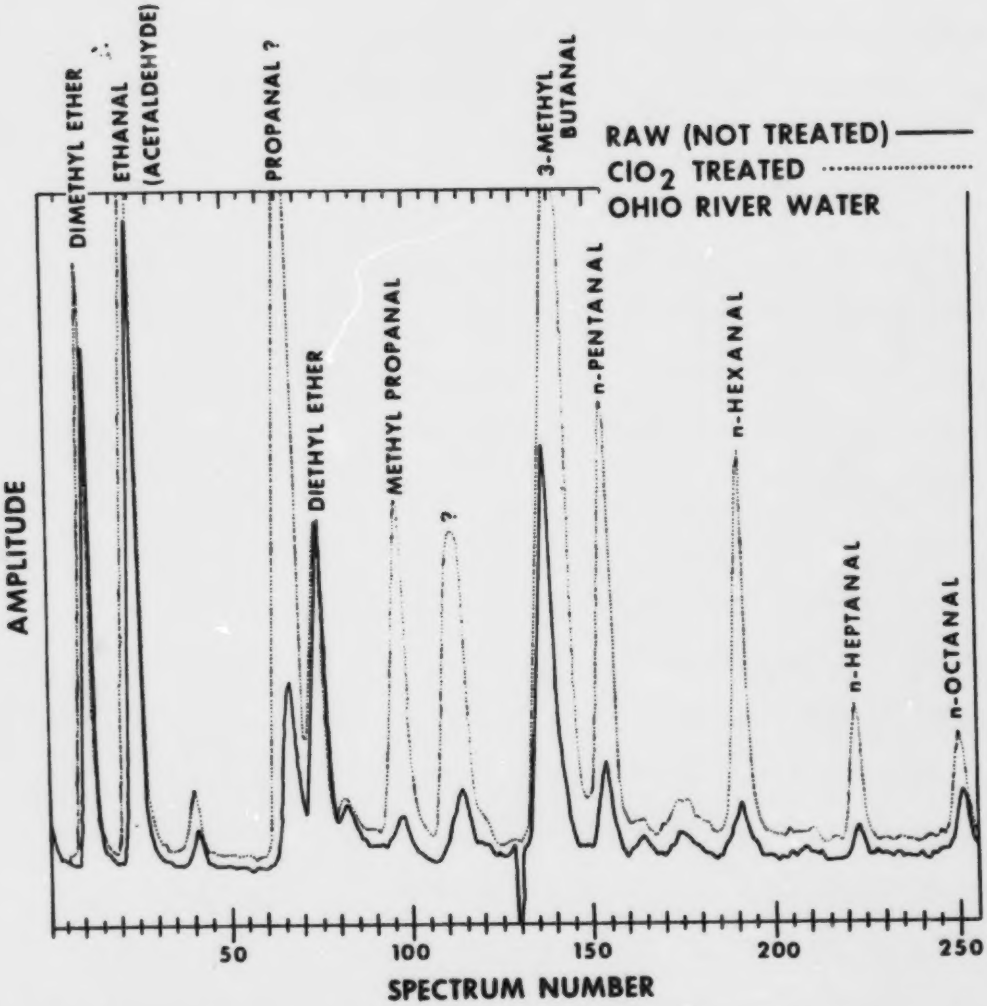


Fig. 16. Extracted ion current profiles (M/E = 29) of raw and chlorine dioxide treated Ohio River water (After Stevens et. al.[84], 1978).

was provided by two references from the food science field.[85,86]

A 1963 reference[85] reported threshold odour levels in water of 0.15 ug/L(ppb) and 0.9 ug/L(ppb) for 3-methyl butanal and 2-methyl propanal respectively. These compounds, among others, were reported, as present in odour from cooked potato products. A 1976 reference reported the odour thresholds in water somewhat higher at 2.0 ug/L(ppb) and 2.3 ug/l(ppb) for 3-methyl butanal and 2-methyl propanal, respectively. They also report an odour threshold of 12.5 ug/L(ppb) for 2-methyl butanal. Clearly, the levels suggested for Edmonton water (a few to tens of ppb of each) would be detectable to the average individual according to these references.

An interesting side issue, was the fact that 36% of their test group (225 total) were insensitive to 2-methylpropanal in that they required, on average, 258 times higher concentration to allow odour detection. The spread of insensitivity was much less (8 to 16 times) for the other compounds. The odour elicited by these compounds was suggested as the primary contributors to the malt odour. They also described the odour of 3-methylbutanal as sweaty.

The odour of 3-methylbutanal (isovaleraldehyde), even as a single compound, is extremely complex. A survey[87] using 140 evaluators at high concnetration (20g/L) used descriptors with the frequency indicated: fruity, other than citrus (4.55%), fragrant (7.58%), aromatic (11.51%), almond

(6.14%),woody, resinous (13.97%), sweet (8.85%), yeasty (4.77%), dirty linen (4.42%), stale (6.5%), musty,earthy, moldy (12.67%), burnt smoky (6.59%), chemical (10.11%), sharp, pungent, acid (18.71%), sour, vinegar (15.82%), oily, fatty (11.15%), paint (6.86%), rancid (16.18%), sweaty (16.32%), sulfidic (5.23%), animal (5.79%), sewer (5.68%), putrid, foul, decayed (17.55%), fecal, manure-like (6.13%), cadaverous, dead animal (4.17%), sickening (26%), dry, powdery (4.42%), light (4.73%), heavy (22.38%), warm (10.21%). Another 88 descriptors were used by at least one of the 140 evaluators. Only one descriptor was used by more than 25% of evaluators, that being "sickening". Clearly with this diverse a response for a single pure chemical, a mixture of at least three of these odorous chemicals, together with chloramines, would easily explain the wide range of responses experienced by Edmontontians.

There is reasonably strong, although still somewhat circumstantial evidence that these branched short chain aldehydes played a major role in the odour incidents of 1985 and 1986. However, prevention of future episodes of this type requires an understanding of how the problem was created. Having only deduced the likely odorous componds after the fact, we can only speculate on their cause and, therefrom, the cure.

We cannot rule out the possbility that these compounds were present in odorous quantities in the raw river water, because they do occur as natural degradation products of amino acids[86] and they have been reported in river samples.[84,88]

City of Edmonton monitoring for volatile organics by gas chromatography is helpful in this reagard. The chromatograms generally show the appropriate peaks in the treated water at times when they were absent in the raw water, but they could have been present in some samples. Unfortunately, we cannot be certain on this issue because the retention times on the packed columns are not discrete enough to distinguish compounds for some of the more complex samples. Spiking studies, analysis with different columns to verify retention times for different chromatography and, preferably, purge and trap GC/MS with fresh samples would be necessary to draw certain conclusions about the presence or extent of these compounds in the raw water relative to the treated water.

If, as seems plausible, the compounds were formed or, at least, enhanced by treatment, the question remains as to how. Stevens' work suggests that chlorine dioxide will produce these compounds from natural waters. That proposition does not specify what starting material is necessary. The usual assumption, in the face of uncertainty, is to attribute the problem to oxidation of humic substances. That may be the answer, but a more specific explanation would be more useful in identifying means for preventing future incidents.

One possbility, following from the observations on the source of the malty odour in foods[86] is that the aldehydes are the oxidation products of specific amino acids. If the oxidant chemical could convert the terminal carbon attached to the carboxyl and amino groups of an appropriate amino acid to a carbonyl group, the appropriate aldehyde would follow (Figure 17).

A recent summary of oxidation byproducts in drinking water treatment[89] provides some interesting observations on the possibilities of these reactions. They note that chlorine dioxide will convert glycine to formaldehyde, which is the equivalent of the reaction necessary to convert the appropriate amino acids into the odorous aldehydes. This work also reports that phenylalanine produces traces of benzylic acid, benzoic acid, mandelic acid and phenylacetaldehyde (benzeneacetaldehyde). Benzoic acid and benzeneacetaldehyde were identified in the extractable organic analyses (Table 10) for both raw and treated samples. The findings on phenylalanine are in direct contradiction of older (1957) work[90] which was cited in a recent (1984) review.[91] The review states that phenylalanine, as well as leucine, the precursor for 3-methylbutanal, and several other amino acids do not react with chorine dioxide under "water treatment conditions". The review misinterpreted the earlier work which was done with 50% acetic acid rather than "water treatment conditions".

Another perspective on possible sources is offered by a French study[92] which considered the occurrence of amino acids in water treatment plants. They found that amino acids are oxidized to the corresponding aldehydes by chlorination and demonstrated that with formation of acetaldehyde from chlorination of alanine (Figure 17). They also found that amino acids were apparently formed in the treatment process. Amino acids may arise from oxidation of peptides and proteins as well as biological activity in the process. An increase in amino acids was noted on the filters perhaps because of biological activites therein.

These findings, along with the realization that amino acids will occur in the raw water and may be produced by any biological activity in the distribution system, presents a much different problem than if the aldehydes are produced from humic materials. The latter can be removed to some degree with coagulation processes while the amino acids will be largely unaffected by such processes. Given this uncertainty, being assured of avoiding future problems of this type will depend upon knowing what conditions will allow formation of the aldehydes. If they can be formed from appropriate amino acids, it will be necessary to understand the occurrence of amino acids at the plants and in the distribution system. Without answers to these questions, we can only suggest some adjustments and hope for the best. The obvious adjustment is to limit the application of chlorine dioxide to post clarification and limit doses in the hope that reduced oxidant combined with reduced humic material will avoid the problem.

Some insight into the chances of solving the problem, without pursuing a better understanding, are provided by a

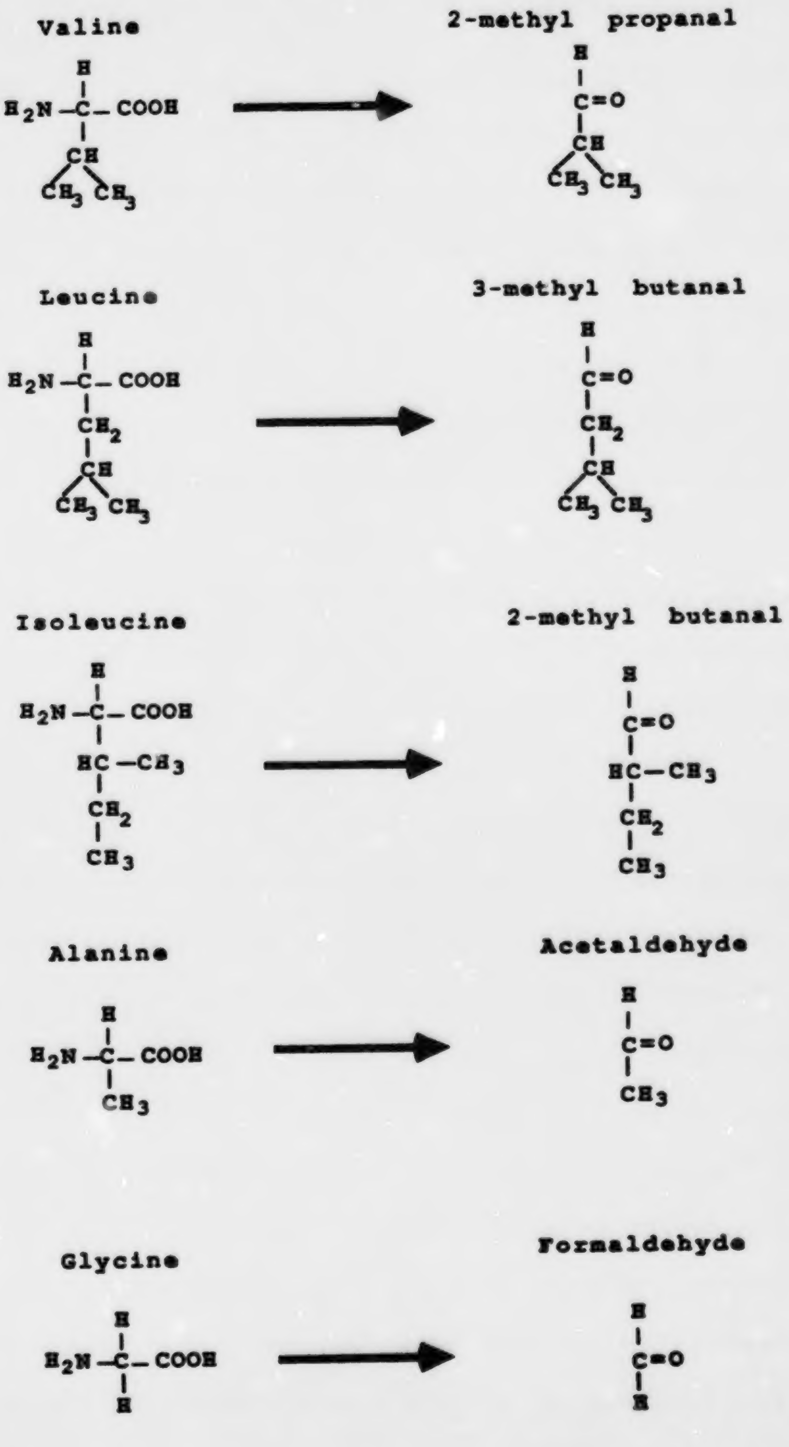


Fig. 17. Oxidation of specific amino acids to aldehydes through conversion of the carboxyl and amino groups to a carbonyl group.

very limited sampling program done at the University of Alberta pilot plant being run by Dr. Huck in support of research into alternate disinfectants (supported by National Health and Welfare and Alberta Environment). This plant is located at the Rossdale site and it draws raw water from the Rossdale intake.

When the dimensions of the taste and odour incident had become apparent, arrangements were made to perform extra threshold odour number (TON) determinations at various points in the pilot plant. These were, raw water, after clarification, filtration and oxidant addition, and after granular activated carbon (GAC) filtration.

Results from this assessment are plotted in Figures 18.1 to 18.3. The raw water TONs on March 4 to 7 were substantially higher than observed in the full scale plant monitoring (Figure 15.2). However, samples were collected at different times so these findings are not necessarily a discrepancy. The treated water TONs for chlorine and chlorine dioxide on March 4 seem to match the peaks at the full scale plants on March 3. They decrease rapidly in all cases as the week progresses. The March 4 data seem to support the observation for the full scale plant with chlorine dioxide, namely, the treated water exhibits a stronger odour than the raw. Apparently, the same result would be expected with free chlorine. However, this pattern was not consistent when TON levels dropped on March 5 and 6. The pattern of treated worse than raw did return on March 7 and 8

Fig. 18.1. **University of Alberta Pilot Plant Threshold Odor Number**

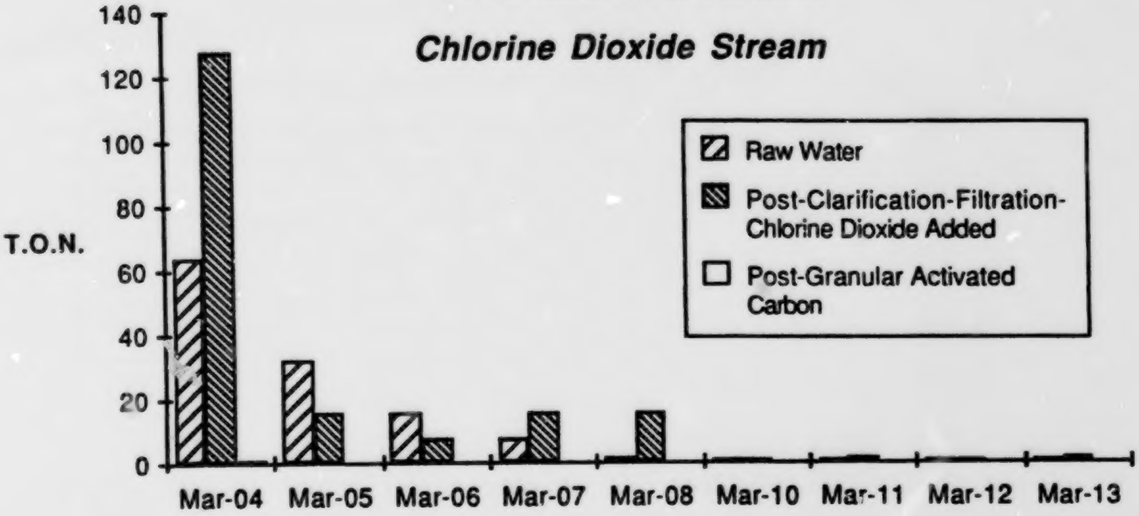


Fig. 18.2. **University of Alberta Pilot Plant Threshold Odour Number**

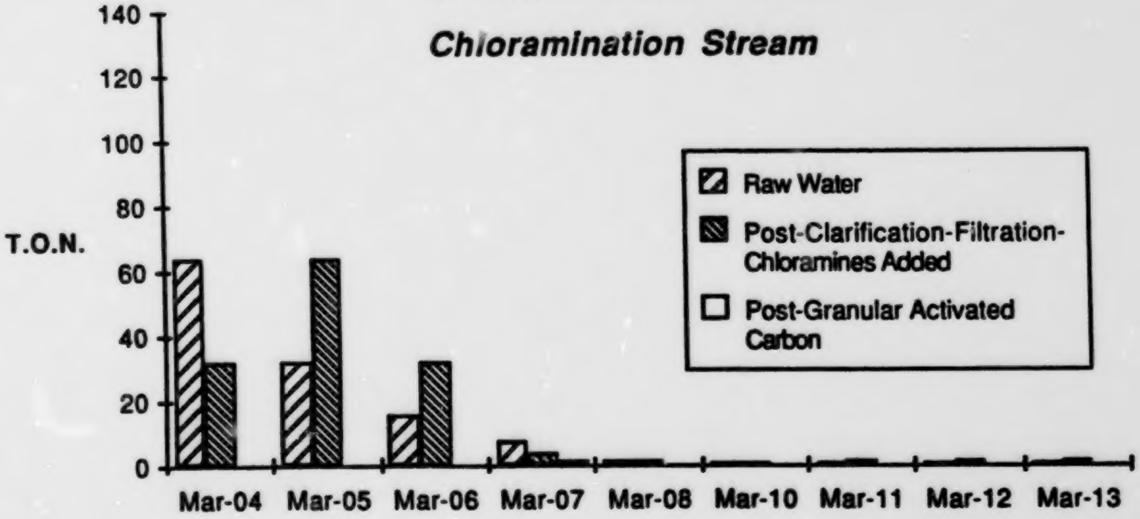
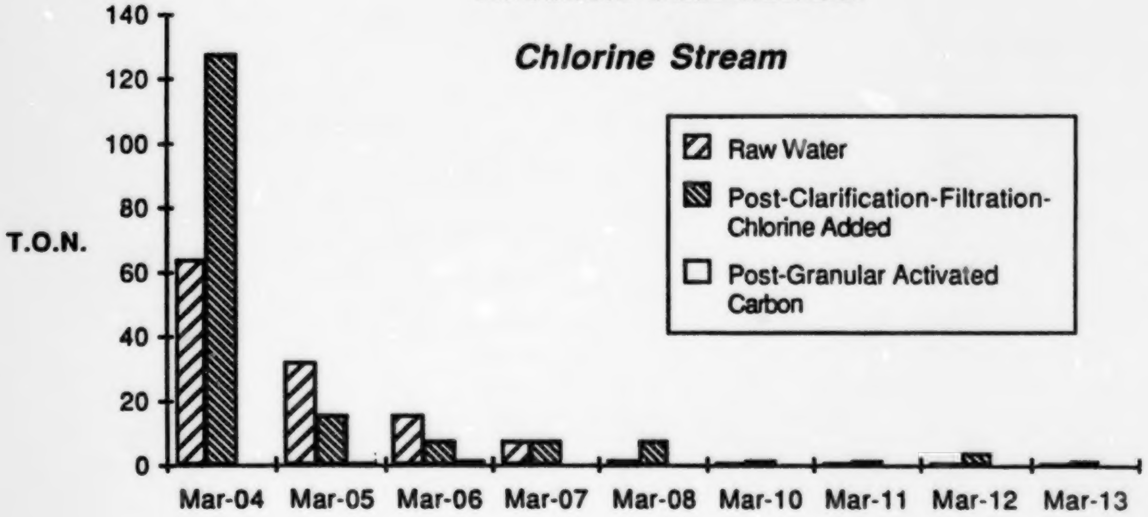


Fig. 18.3. **University of Alberta Pilot Plant**

Threshold Odor Number



The results with chloramines are also hard to interpret. They show a lower odour for treated than raw on March 4 but then they switch and show higher odour on March 5 and 6. There is no obvious explanation for these variations, other than mislabelling of the samples, an explanation that cannot be verified or disproven.

One factor which did seem to be consistent was the ability of granular activated carbon adsorption to successfully remove odour. That option, at least, provides a fall back position if inexpensive means for odour control cannot be demonstrated.

3.6 Disinfection at Edmonton Water Treatment Plants

3.6.1 Recent and Past Practice

The provision of safe drinking water has always depended upon measures to control the presence of microorganisms to insure that water cannot harbour disease causing organisms in circumstances which can initiate infection. Such assurance requires the use of a disinfection process as an integral part of the water treatment process scheme. The practical disinfection options currently available to water utilities are limited.

Four disinfectants are commonly in use, namely: free chlorine, chloramines, ozone and chlorine dioxide. All of these chemicals are oxidants which means that each has the ability to accept electrons. That ability allows the oxidant to participate in reactions which can lead to partial or complete destruction of organic chemicals. The former is much more likely than the latter under the mild reaction conditions which are feasible in water treatment processes.

This disruptive capability allows oxidant chemicals to inactivate or disinfect microorganisms. The more powerful that oxidant capability, the more reactive the disinfectant. In recent years, we have become aware that disinfection chemicals inevitably produce reaction byproducts in natural waters. The first disinfection byproduct to raise major toxicology concerns was chloroform which is produced in the chlorination of aquatic humic substances.[47]

Since that discovery in 1974, there has been considerable research attention directed towards the nature and health concerns of disinfectant byproducts. A review of all these issues is well beyond the scope of the current discussion. The key point is that there is currently no known disinfectant which can perform its function without producing any byproducts.

As noted by Dr. Bull, in his expert report in this Volume, very few of the compounds which can be formed from disinfectant reactions with organic chemicals, naturally or otherwise present in the water, have been subjected to toxicological testing. This gap arises because these chemicals are generally not used commercially.

Because of this complex situation, some researchers have chosen to assess the combination of substances which may be formed by means of general measures such as mutagenicity.

This approach has both strengths and weaknesses, as noted by Dr. Bull.

We must accept a need to disinfect water or we will certainly cause illness in the population. Experience has, and still is, verifying this reality all too frequently in underdeveloped regions of the world. Against the certainty of illness that will occur with inadequate disinfection must be weighed the risks which go with any particular disinfectant chemical.

The safety of the consumer will be maximized when an appropriate balance is achieved between the competing concerns. Hence, disinfection cannot be pursued without concern for the chemical consequences but, likewise, disinfection cannot be significantly compromised based only upon concerns about "potential" health effects of chemical byproducts. Some tradeoff with these competing objectives will likely be necessary.

Edmonton has used chloramines as primary disinfectants since 1949. These are formed by adding ammonia to the water before applying chlorine. The latter readily reacts with ammonia to form mono- and dichloramines. These chemicals have less disinfecting power than free chlorine but they form a more stable residual which will persist in the distribution system. Since the discovery that chloroform and other trihalomethanes are produced by free chlorine, chloramines have received more interest because they do not form trihalomethanes to any appreciable degree. The major negative factor concerning the chloramines are the questions raised about the adequacy of their disinfection capability (see Logsdon, Payment and Tobin reports, this Volume).

Chlorine dioxide had been used intermittently on a small scale by Edmonton, but in 1982, high yield generators were installed on plants 1,2 and 4 to evaluate this chemical for taste and odor control during spring runoff. The premise of using an oxidant for taste and odor control is that the odorous substances can be chemically modified to non-odorous compounds or totally converted to mineral endproducts. As noted in the introduction, the latter is unlikely in most cases, for realistic reaction conditions in water, particularly if significant quantities of extraneous organic matter are present to provide an oxidant demand. Hence, oxidative control of taste and odor chemicals must be approached with the knowledge that chemical byproducts will be produced. These may or may not be odorous but care should be exercised in trading a known problem, i.e. an odorous compound which may possess low toxicity, for byproduct compounds of unknown toxicity but no odor.

Although chlorine dioxide was acquired for the purposes of taste and odor control under runoff conditions, the *Giardia* outbreak of 1982/83, lead to a concern about the adequacy of chloramines as a disinfectant for *Giardia* cysts. To assure an additional barrier to *Giardia*, the City decided to opt for year-round application of chlorine dioxide in

November 1983. Chlorine dioxide was not installed on plant 3 until 1984.

With the intention of using chlorine dioxide as a disinfectant, the City developed an operating guide of providing sufficent dosage of chlorine dioxide to maintain a residual of 0.10 to 0.19 mg/L. These numbers were based upon the lowest levels which could be reliably analyzed by techniques in use.

Control of dosage to maintain an adequate residual is necessary if chlorine dioxide is the primary means of protection against pathogens. Dr. Logsdon has discussed the needs for an adequate residual and contact time for *Giardia* inactivation (see Logdson report, this Volume). The problem arises when the dissolved organic carbon content of the raw water rises and creates a rising demand for the chlorine dioxide. Then, rather than producing a residual of chlorine dioxide which can achieve disinfection, the oxidant can be totally consumed in extraneous reactions which produce chemical byproducts. As well, 50% of the chlorine dioxide dose will typically appear as residual chlorite when reacting with humic materials in water.[93,94] This chemical is still an oxidant, but it has no significant disinfectant properties. Unfortunately, it does have toxic effects, at sufficiently high doses. These concerns are discussed by Dr. Bull (see Bull report, this Volume).

In summary, a practice of dosing chlorine dioxide to the limits of generator capacity, in search of a dose adequate to sustain a measurable residual will, in the face of a raw water high in dissolved organic matter, create both organic byproducts and high levels of chlorite. This appears to have happened in the spring of 1985 and again in the spring of 1986 before the objective for chlorine dioxide dosage was changed to limit the combined residual of chlorine dioxide plus chlorite to 0.5 mg/L. Under high demand conditions, that limit would be expected to consist entirely of chlorite and there would likely be no effective residual of chlorine dioxide to act as a disinfectant.

Under the previous policy of dosing to meet demand, some very high doses were used. They peaked at 2.75 mg/L of chlorine dioxide in March 1986 and 3.6 mg/L in September 1985. In the past, there has been no requirement to monitor for chlorite because this chemical is not covered by the Canadian Drinking Water Guidelines.[10] However, calculations done on analyses previously performed for measuring chlorine dioxide residual have shown that values for single grab samples from reservoirs reached 1.0 mg/L of chlorite in September 1985 and values as high as 1.35 mg/L of chlorite were reached in April 1985.

Guidelines for controlling chlorine dioxide dosage have not been widely published. The U.S. Environmental Protection Agency[95] specified a limit of 1.0 mg/L in 1978, but their wording was ambiguous:

"Studies with cats have shown that chlorite has a deleterious effect on red blood cell survival rate at chlorine dioxide concentrations above 10 mg/L. Therefore, a limit of 1.0 mg/L is necessary to prevent possible adverse effects on sensitive individuals, particularly children. Additional studies are underway to clarify this matter."

This does not make it clear if they are recommending a dosage limit of 1.0 mg/L of chlorine dioxide or a chlorite residual limit of 1.0 mg/L. In light of current knowledge, a residual limit of 1.0 mg/L chlorite seems less likely than a dosage limit on the chlorine dioxide.

Although Dr. Bull has not specified a maximum acceptable number for chlorite which he feels we must adopt, he did demonstrate a calculation, using a safety factor of 100 from experimental dosages, which would yield an objective level of 0.1 mg/L of chlorite.

Clearly, the apparent high levels reached in 1985 have infringed significantly on the safety factor of 100. Such levels should not be accepted in future practice. The current practice of limiting combined chlorite plus chlorine dioxide to less than 0.5 mg/L has been an acceptable interim measure.

The joint Federal-Provincial Drinking Water Committee has placed the question of guideline limits for chlorite, chlorate and chlorine dioxide on its agenda so some further guidance should be forthcoming.

In the mean time, we are left with a problem of finding the balance which was mentioned at the outset. There should be no difficulty in substantially limiting the dose of chlorine dioxide which is used primarily for taste and odor control. The benefits of chlorine dioxide for this purpose have not been rigorously demonstrated because most observations of success here and elsewhere seem to lack any controlled comparisons. Even if there were hard evidence to show that chlorine dioxide could substantially control Edmonton's taste and odor problems, I would not like to see resulting chlorite residuals for strictly an aesthetic control measure infringe upon Dr. Bull's safety factor of 100.

If however, there is demonstrable need for chlorine dioxide to protect Edmontonians from the occurrence of pathogens, such as *Giardia*, higher dosages may be justified. That justification needs to arise from a demonstration that the raw water may contain *Giardia* or viruses (of similar concern) and that the plants may have difficulty in resisting a pathogen challenge. Unfortunately, monitoring of water for *Giardia* and viruses has proven difficult and achieving negative results does not guarantee pathogen absence. However, given that: *Giardia* is endemic in the population, cysts are discharged in large numbers in the feces from infected hosts and the existence of combined sewer overflows and sanitary sewage cross connections discharging upstream from Rossdale, an assumption of potential *Giardia* challenge at Rossdale seems reasonable. That challenge need not be limited to runoff conditions because the sanitary sewage flows arising from cross connections would be independent of storm flows.

The second condition for demonstrable chlorine dioxide need was a concern whether the treatment plants could respond when challenged by a pathogen. In reviewing the Rossdale plant in 1983, after the *Giardia* outbreak, Lippy[96] concluded that: "under challenge, the plant could not respond effectively to remove or inactivate *Giardia* cysts." He based his assessment upon inadequate turbidity removal and dependence only upon chloramination with inadequate contact time for disinfection. The adequacy of turbidity removal has again been questioned by Dr. Logsdon (Logsdon report, this Volume). Some improvement in chloramine disinfection will arise from use of a new 50 ML clear water reservoir which could provide up to 8 hours of disinfectant contact time before water would arrive at a consumer's tap. Actual contact times will vary because the reservoir may not be maintained full during high water demand conditions and there is likely to be some short circuiting. At the time of the *Giardia* outbreak, water leaving the plant could have arrived at the first consumer within minutes, with travel time determined by distance from the plant. Hence, consumers closer to the plant would be afforded less disinfectant protection than distant consumers.

The situation at the E.L. Smith plant does not seem to pose the same risk of *Giardia* exposure. There is concern with *Giardia* arising from beavers upstream but the reality of this risk remains to be documented. Certainly, the risk from human fecal contamination of raw water at Smith is about as low as is likely to be found for a river supply serving a city the size of Edmonton. The Smith plant also has a 125 ML clear water reservoir (in 3 cells) which could provide 12-20 hour of contact time for chloramines to inactivate *Giardia* cysts. These theoretical contact times would be subject to the same limitations noted above for Rossdale.

3.6.2 Recommendations for Future Practice

Considering the situation as it now exists, there is no magic "quick fix" which will solve all problems, yet have no associated negative features. However, by considering several factors together, a substantial margin of disinfection safety should be achievable without substantially compromising concerns about disinfectant byproducts. The necessary components cannot be considered in isolation if they are to achieve the desired overall benefit. The following recommendations include some actions which are already under way but all components will be listed to assure that the complete package is viewed in an integrated fashion. These recommendations have been developed with the philosophy that one should not embark on radical changes in practice without substantial prior knowledge or experience to sugggest that the new option will not create more problems than may currently exist.

1. The intake for the Rossdale plant must be moved upstream to a point where water quality equivalent to that experienced at the E.L. Smith plant can be reliably obtained.

Preliminary modelling calculations by Dr. Smith and associates (see Smith report, this Volume) indicate that it may not be necessary to move all the way upriver to the E.L.Smith cross-section to achieve that goal.

2. The point of chlorine dioxide addition used when river dissolved organic carbon concentrations begin to exceed about 4 to 5 mg/L must be downstream of alum coagulation and clarification. This is necessary to obtain the benefit of removing raw water organic load by clarification before undesirable reactions with the oxidant can occur. Because the foregoing levels are somewhat arbitrary, process development work is needed to experimentally determine maximum levels of raw water organic carbon which can be tolerated without exerting substantial short term demand.I would interpret substantial as being greater than about 50% of dose being wasted on demand rather than disinfection.

Application of chlorine dioxide to raw water for algae control at Rossdale may still be possible without undesirable byproduct formation when raw water organic carbon levels remain below the criterion determined. The need for that application should be reduced by the building of structures for heat retention over the clarifiers and given water from an upstream intake which will be less nutrient-rich than the current Rossdale supply. Moving ammonia addition downstream would likely also help reduce algae growth in the clarifiers.

3. Under no circumstance should chlorine dioxide be added simultaneously with powdered activated carbon as has been practice in the past. The carbon will exert a demand upon the chlorine dioxide with concurrent wastage of the feed chemical (sodium chlorite). It is not clear whether chlorine dioxide consumed in this way could appear as chlorite. As well, there is the possibility of the carbon catalyzing oxidation reactions at the carbon surface leading to further unknown byproducts.

4. The point of chlorine dioxide addition must be provided with rapid mixing. Otherwise, very high concentrations of chlorine dioxide can be sustained in contact with organic matter in the water leading to the possiblity of forming many unknown byproducts which would not be formed at fully mixed dosage levels.

5. The coagulation and flocculation processes must be optimized for removal of raw water organic carbon. This may entail the use of supplementary or alternative coagulants, pH adjustments or more major changes in process sequence (see Bernhardt report, this Volume). Process optimizaton requires detailed bench and pilot scale investigations which requires a substantial commitment with highly qualified manpower and laboratory support. This must be an ongoing commitment to deal with new problems and new process developments.

6. The entire process must be optimized with an aim of pursuing the turbidity goal of 0.1 NTU proposed by Dr. Logsdon and others. Although that goal may entail a long term program given existing equipment and water demand constraints, the implications to the disinfection dilemma are profound. The critical pathogens for disinfection concern are *Giardia* cysts followed by enteric viruses. Both of these pathogen classes are effectively removed along with inanimate matter which causes turbidity. Thus, although the current license limit of 1 NTU derived from the Canadian Drinking Water Guidelines[10] is certainly adequate for aesthetic concerns and assurance of adequate bacterial disinfection, this level does not optimize the physical process for *Giardia* or virus removal.

Such optimization, although challenging and possibly difficult, does not carry an attendant risk associated with greater oxidant chemical use. Furthermore because most metals and hydrophobic organic pollutants will be associated with suspended particles, optimized turbidity removal can increase safety factors for exposures to such elements or compounds of health concern. Finally, turbidity reduction will reduce disinfectant demand in the distribution system which can further limit demands for disinfectant dosage.

This goal should, in the long term, be pursued for all water conditions. Such improvements may entail the addition of more treatment stages upstream of filtration because filter capability for production of low turbidity effluent will be limited by the turbidity of the water applied

7.The need for continued practice of chorine dioxide disinfection at E.L.Smith needs to be demonstrated. Based on fecal coliform levels in the raw water and upstream geography, there is little apparent risk from pathogens of human origin. Without convincing evidence of a real risk posed by *Giardia* originating with upstream wildlife, there is no other apparent justification for chlorine dioxide use at the E.L.Smith plant. The likelihood of taste and odor problems which can be solved by chlorine dioxide has not been documented at the E.L.Smith plant. Even if it were, dosages for control of an aesthetic problem should be much stricter (chlorite residual of about 0.1 mg/L) and there would be no need for year-round application. The City of Edmonton and Alberta Environment should jointly embark on a study to initially review the likely *Giardia* risk for E.L.Smith, and depending on the clarity of the initial findings, embark on sufficient monitoring with the best available methods to document the extent of existing risk. Without demonstrable need of chlorine dioxide for disinfection, the cost and unnecessary addition of chlorine dioxide byproducts to the water can and should be avoided.

8. The risk assessment approach should be used to assist the development of a site specific disinfection approach for Edmonton. Given the many variables, including: pathogen resistance, contact time, disinfectant demand, water temperature and water chemistry, Edmonton needs to develop its own specific rationale for selecting disinfectant type, residual concentration and contact time. That rationale should be experimentally derived and verified.

9. The foregoing recommendation for the E.L.Smith plant clearly has implications for Rossdale, once it receives raw water of equivalent quality. The same principles should apply at that time.

10. In the interim, chlorine dioxide use carries a responsibility to maintain careful control over generator efficiency, dosage levels and measured residuals. That responsibility requires a high level of analytical competence which can only be assured by direction from highly trained analysts. This is an area which requires graduate level training in chemistry (preferably analytical chemistry) with an appropriate degree of experience. The City has reached a point in its analytical control of chlorine dioxide generation and experience which is much superior to many other users of chlorine dioxide. However, this has only been recently achieved and was not the case when chlorine dioxide use was implemented for year-round use in 1983. Continued use of chlorine dioxide requires that the current level of analytical control be maintained and expanded as better techniques become available. Alberta Environment should engage the Alberta Environmental Center to cooperate with Edmonton in developing further analytical improvements. Of course, such methods development should also result in inclusion of chlorite and chlorine dioxide in the Alberta Environment Treated Water Surveys for communities using chlorine dioxide.

11. Use of chlorine dioxide should continue to be restricted to dosages which will produce a maximum residual of 0.5 mg/L of the combination of chlorine dioxide plus chlorite. This, however, must be viewed as an interim measure until the Rossdale intake can be moved in accordance with recommendation 1. This level must also be viewed as a true maximum. Review of records of past dosage levels suggests that a target of less than 0.3 mg/L is probably realistic for normal operating conditions.

12. Chlorine dioxide application should be cross-checked by mass balance controls on sodium chlorite consumption, much as are currently used for fluoride control. Operators can assure the maximum possible dose of chlorine dioxide is limited to set levels by checking the mass quantities of raw material fed to the generators.

13. The City, as part of its process development commitment should carefully follow developments with ozone applications. Ozone may be useful in Edmonton if the evaluation of upstream *Giardia* risk confirms the long term need for a disinfectant additional to chloramines. Ozone should not, however, be considered simply as a strong oxidant for destroying high levels of dissolved organic carbon as had been practiced with chlorine dioxide. Similar problems of byproduct formation could be expected.

There must be a realization that use of a strong oxidant for conversion of organics cannot be used in isolation. Applications of ozone for organics destruction elsewhere have been integrated with biological activated carbon. Some form of organics removal, such as granular activated carbon, must be provided if ozone or other strong oxidants are to be applied to waters containing significant (more than a few mg/L) dissolved organic carbon.

14. The city should adopt a long term goal of maximizing the biological stability of treated water. By minimizing the presence of available organic substrate for downstream biological activity, the disinfectant demand in the distribution system can be minimized, thereby allowing minimization of disinfectant dose.

4. CONCLUSIONS

4.1 Raw Water Quality

1. The Alberta Surface Water Quality Objectives (ASWQO)are quite comparable to water quality criteria used by other jurisdictions. In a few cases ASWQO levels were either not provided or were higher than values used by others. In these cases, a review of the ASWQO has been recommended and in specific cases lower values are likely required. In other cases, the ASWQO were lower than values used by other jurisdictions often reflecting the multiple use nature of the ASWQO.

2.An updating of the ASWQO is necessary to make it consistent with current analytical procedures.

3.For the majority of parameters, typical North Saskatchewan River water quality coming into Edmonton is good relative to the Alberta Surface Water Quality Objectives or alternate objectives from other jurisdictions where the latter are lower. Some individual results for metals warranted discussion but there were no cases other than phenolics and turbidity where average water quality approached or exceeded the objectives.

4. Based upon review of water quality monitoring dating back to 1963 at four sites upstream and in the city, their is no evidence of any trends towards decreasing upstream water quality.

5. Overall, the North Saskatchewan River compares favorably as a raw water source with the locations chosen for comparison and described for treatment practice in the expert reports. All sources compare favourably with the Canadian Drinking Water Guidelines.[10] The raw water sources chosen for comparison range from fair to very good. By comparison the North Saskatchewan River would rate as a good overall raw water source. This rating must acknowledge periodic conditions of high turbidity, color and dissolved organic carbon which will challenge effective treatment.

6. There are very few industrial or municipal point source discharges to the North Saskatchewan River upstream of Edmonton. Given the current situation, the river upstream of Edmonton should be regarded as an essentially undeveloped watershed

7. Urban runoff within the city limits has a significant deleterious impact on the raw water quality for the Rossdale plant, particularly with regard to microorganisms (see Smith report, this Volume) and hydrocarbons. Contributions from snow dumps are not as well known despite major water quality monitoring programs since 1973. These have focussed on metals and have not provided convincing data on hydrocarbons.

8.There is no evidence of any significant impact of agricultural chemical use upstream of Edmonton upon raw water source quality.

9. Natural organic carbon, in the form of humic materials and products of vegetation decay, are contributed to the North Saskatchewan River by several tributaries draining muskeg and parkland areas. These impacts are most

noticeable in the spring but can continue in a measureable way year-round.

10. The spring 1986 monitoring program clearly documented the degraded water quality experienced at Rossdale relative to that at E.L.Smith. This impact, most noticeable with hydrocarbons, was attributable to urban runoff experienced mainly during the last week of February.

11. Upstream rural runoff, as evidenced by high dissolved organic carbon, arrived on March 1, 1986 and impacted both the E.L.Smith and Rossdale plants for about a week. The bulk of this organic matter does report in the extractable organics fraction analyzed by gas chromatogrpahy.

12. The spring 1986 monitoring program documented the occurrence of two types of runoff. The urban runoff affects the Rossdale plant exclusively while the rural runoff affects both Smith and Rossdale almost equally.

13. The Rossdale intake is highly vulnerable to chemical spills arising from the storm sewer network. This situation does not provide an adequate margin of safety for a public water supply.

14. The E.L.Smith intake is far less vulnerable given the limited degree of upstream activity. A major spill event would be necessary to affect the center of the river and the upstream location of this intake.

4.1 Treated Water Quality

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1. Treatment plant performance, as reflected in City reports, has increased substantially since 1981, when reporting performance was inadequate for a utility of this size.

2. In recent years, the plants have demonstrated a high degree of compliance with their Clean Water Act license requirements.

3. Review of the Alberta Treated Water Survey data, collected independently by Alberta Environment from the distribution system, confirms the compliance record reported by the City.

4. Comparison of treated water quality for several health-related parameters with Calgary and other cities in Canada, the United States and Europe indicates that Edmonton water is substantially equivalent in quality, on a routine basis. Spring runoff odour events and hydrocarbon impacts on Rossdale are not represented in these comparisons.

5. The Alberta Treated Water surveys provide a very substantial data base, moreso than any available from other locations surveyed, on the occurrence of trace organic contaminants in Edmonton water. This data base indicates that trace organics occur only in low concentrations with limited frequency under normal water conditions. Urban runoff impact on Rossdale can negatively influence that situation.

6. Taste and odour events have commonly occurred in the past. They have been blamed on a variety of causes, but substantial evidence of causation has been lacking.

7. There is evidence that the 1985 and 1986 spring odour events have been caused by the presence of three odourous, short chain aldehydes: 2-methyl propanal, 2-methyl butanal and 3-methyl butanal. The specific origin of these chemicals is still subject to speculation. Avoiding their re-occurrence in the future cannot be assured without specific investigations aimed at determining their likely source. Such studies need not wait until next spring.

8. Apart from odor control, the only substantial water quality issue relates to current disinfection practice. Concern for the health implications of disinfectant byproducts and for adequate disinfection to protect the water supply from *Giardia* cysts requires the implementation of the specific recommendations provided in Section 3.6.2.

REFERENCES

1. Inland Waters Directorate. 1983. Historical Streamflow Summary, Alberta, 1982.Water Resources Branch, Water Survey of Canada, Ottawa.

2. Stanley Associates Engineering Ltd. 1975. North Saskatchewan River Water Quality Study. Prepared for Alberta Environment, by Stanley Associates Engineering Ltd, Edmonton.

3. Water Quality Branch. 1977. Alberta Surface Water Quality Objectives. Alberta Environment, Standards and Approvals Division, Edmonton.

4 CCREM. 1985. Inventory of Water Quality Guidelines and Objectives 1984. Prepared for the Canadian Council of Resource and Environment Ministers, by the CCREM Task Force on Water Quality Guidelines.

5. Department of Environment and Workplace Safety and Health. Surface Water Quality Management Proposal. Volume 1: Surface Water Quality Objectives. Water Standards and Studies Report No. 83-2. Manitoba.

6. Ministere de l'Environment. Inventaire des Criteres de Qualite. 1984. Service de la qualite de eaux. Quebec.

7. Environment Canada. Guidelines for Surface Water Quality. Volume 1: Inorganic Chemical Substances. Volume 2: Organic Chemical Substances. 1979-83. Inland Waters Directorate. Water Quality Branch. Ottawa, Canada.

8. U.S. EPA. 1976. Quality Criteria for Water. U.S. Environmental Protection Agency, Washington, D.C., U.S.A.

9. U.S. EPA. 1980. U.S. Environmental Protection Agency, Water Quality Criteria Documents. EPA-440/5-80-015 to 079. Also in the Federal register, Part V. Friday, November 28, 1980. **45**(231).

10. Health and Welfare Canada. 1978. Guidelines for Canadian Drinking Water Quality. Prepared by the Federal-Provincial working group on drinking water of the Federal-Provincial committee on environmental and occupational health, National Health and Welfare. Canadian Government Publishing Center, Supply and Services, Hull, Quebec.

11. U.S. EPA. 1985. Federal Register. U.S. Environmental Protection Agency. Part IV. Wednesday, November 13, 1985. **50**(219).

12. American Public Health Association. 1985. Standard Methods - For the Examination of Water and Wastewater (sixteenth edition). American Public Health Association, Washington, D.C.

13. Verschueren. 1977. Handbook of Environmental Data on Organic Chemicals. Van Nostrand Reinhold Company, New York.

14. Alberta Environment. 1986. Naquadat Detailed Report, Surface water data. Historical Data Relating to the North Saskatchewan River. Alberta Environment, Water Quality Branch, Edmonton.

15. Bouthillier, P.H. 1984. A History of Stream Pollution Assessment and Control - North Saskatchewan River, 1950's to 1980's. Prepared for Alberta Environment, Edmonton.

16. Reynoldson, T.B. 1983. North Saskatchewan River Water Quality, 1970 - 1981. Alberta Environment, Water Quality Branch, Edmonton.

17. Inland Waters Directorate. 1975. Water Quality Data, Alberta, 1961-1973. Environment Canada. Water Quality Branch, Ottawa, Canada.

18. Alberta Environment. 1975. North Saskatchewan River Water Quality Data, 1973-1975. Alberta Environment, Environmental Protection Services, Edmonton.

19. Inland Waters Directorate. 1980. Detailed Surface Water Quality Data, Alberta, 1974-1976. Environment Canada. Western and Northern Region Water Quality Branch, Calgary, Alberta.

20. Inland Waters Directorate. 1982. Detailed Surface Water Quality Data, Alberta, 1977-1979. Environment Canada. Western and Northern Region Water Quality Branch, Calgary, Alberta.

21. Inland Waters Directorate. 1984. Detailed Surface Water Quality Data, Alberta, 1980-1981. Environment Canada. Western and Northern Region Water Quality Branch, Calgary, Alberta.

22. Inland Waters Directorate. 1985. Detailed Surface Water Quality Data, Alberta, 1982. Environment Canada. Western and Northern Region Water Quality Branch, Regina, Saskatchewan.

23. Slobodian, S. 1985, 1984. North Saskatchewan River Water Quality Monitoring Program, 1983, 1984 Final Reports. City of Edmonton, Transportation Department, Engineering Division.

24. City of Ottawa. 1985. Physical, Chemical and Bacteriological Characteristics of Ottawa River Water, Raw and Treated, Ottawa, Ontario. Analytical results taken during 1985 at the Britannia and Lemieux Island water purification plants, Ontario.

25. City of Brantford. 1985. Physical and Chemical Characteristics of the Grand River Water, Raw and Treated Brantford, Ontario. Analytical results taken during 1985 at the Brantford water purification plant, Ontario.

26. Huck, P.M. 1986. Raw and finished water quality Lengg Plant, Zurich, Switzerland (1985 data). (In) Evaluation of Advanced European Drinking Water Treatment in Relation to Practice at Edmonton. P.M. Huck, (this volume), Edmonton, Alberta.

27. Huck, P.M. 1986. Raw and finished water quality Langenau, West Germany (1985 data). (In) Evaluation of Advanced European Drinking Water Treatment in Relation to Practice at Edmonton. P.M. Huck, (this volume), Edmonton, Alberta.

28. St. Louis County. 1985. Table 2-1, St. Louis County Water Company Average Analysis of Finished Water Based Upon Daily Samples Collected at Central Plants 1 & 2, 1984. St. Louis County Water Company, Missouri.

29. City of Edmonton. 1986. Prevention of Run-off from the Combined Sewer System from Going into the North Saskatchewan River. Memorandum of Conveyance to the Office of the City Manager. City of Edmonton, Alberta.

30. City of Edmonton. 1984. Water Treatment Plants 1984 Annual Report. Water and Sanitation, Water Treatment Section. City of Edmonton, Alberta.

31. Alexander, H.C., W.M. McCarty, E.A. Bartlett, and A.N. Syverud. 1982. Aqueous Odor and Taste Threshold Values of Industrial Chemicals. *J. Am.Water Works Assoc.* **74**:595-599.

32. Reynoldson, T.B., and S. Livingstone. 1983. Effects of Spring Runoff on the North Saskatchewan River in the City of Edmonton. Water Quality Control Branch, Alberta Environment, Edmonton.

33. Livingstone, J.S. 1986. Water Quality Data for the North Saskatchewan River - Raw Data 1983. Provided by the Water Quality Control Branch, Alberta Environment, Edmonton.

34. Kinetic Environmental Research Laboratories Ltd. 1982. GC/MS Data on Water Samples Taken at the E.L. Smith Plant (March 29, April 15, and April 23, 1982), and the Rossdale Plant (March 29, April 15, and April 23, 1982). Kinetic Environmental Research Laboratories Ltd. Nisku, Alberta.

35. Anonymous. 1983. Enclosure II - What we are trying to treat. City of Edmonton, Alberta.

36. Enviro-Test. 1984. Chemical Analysis Report. Prepared for Edmonton Water and Sanitation, City of Edmonton, by Enviro-Test Laboratories, Edmonton.

37. City of Edmonton. 1983. Water Treatment Plants 1983 Annual Report. Water and Sanitation, Water Treatment Section, City of Edmonton, Alberta.

38. City of Edmonton. 1985. Report on Storm Sewer Discharges - Summary of Data for Period 1980-1984. Prepared by Wastewater Treatment, Edmonton Water and Sanitation, City of Edmonton, Alberta.

39. Slobodian, S. 1982. Environmental testing program, snow disposal operations, 1973-1982. Interim Report. City of Edmonton, Transportation Department, Engineering Division.

40. Gummer, W.D. 1979. Pesticide Monitoring in Prairies of Western Canada. Water Quality Interpretive Report #4. Environment Canada, Inland Waters Directorate, Water Quality Branch, Western and Northern Region. **En 36-51814.** 14 pp.

41. Aquatic Biology Group. 1984. Pesticide and PCB levels in fish from Alberta. *Chemosphere* **13**(1):19-32.

42. Alberta Environmental Centre. 1984. Methoxychlor and 2,2-bis(p-methoxyphenyl)-1, 1-dichloroethylene Residues in Fish in Alberta. Alberta Environmental Centre, Vegreville. AECV84-R1:24 pp.

43. Alberta Environmental Centre. 1984. Partitioning of Polychlorinated Biphenyls in the North Saskatchewan River. Alberta Environmental Centre, Vegreville. AECV84-R3:73 pp.

44. Ongley, E.D., D.A. Birkholz, J. Carey, and M.R. Samoiloff. 1985. Occurrence and Pathways of Toxic Chemicals in the North Saskatchewan River. National Water Research Institute. Inland Waters Directorate. Environment Canada, Winnipeg. NWRI Contribution #85-107.

45. Alberta Environmental Centre. 1983. Chemical Residues in Fish Tissues in Alberta II. Mercury in the North Saskatchewan River. Alberta Environmental Centre, Vegreville. AECV83-R2.

46. Gjessing, E.T. 1976. Physical and Chemical Characteristics of Aquatic Humus. Ann Arbor Science Publishers. Ann Arbor, Mich.

47. Rook, J.J. 1974. Formation of Haloforms During Chlorination of Natural Water. *Water Treatment and Examination*. 23:234-243.

48. Enviro-Test. 1986. Chemical Analysis Report - June 10, 1986. Prepared for the City of Edmonton by Enviro-Test Laboratories, Edmonton.

49. Anonymous. 1984. Spill Summary 1981-1984. Edmonton Water and Sanitation, City of Edmonton, Alberta.

50. Masuda, A. 1979. Investigation of Tainted Water City of Edmonton Water Supply - October, 1979. Water Quality Control Branch, Alberta Environment, Edmonton.

51. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of January. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

52. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of February. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

53. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of March. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

54. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of April. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

55. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of May. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

DRAFT

56. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of June. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

57. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of July. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

58. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of August. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

59. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of September. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

60. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of October. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

61. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of November. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

62. City of Edmonton. 1985. Water Treatment Plants Monthly Report - 1985, Month of December. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

63. City of Edmonton. 1986. Water Treatment Plants Monthly Report - 1986, Month of January. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

64. City of Edmonton. 1986. Water Treatment Plants Monthly Report - 1986, Month of February. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

65. City of Edmonton. 1986. Water Treatment Plants Monthly Report - 1986, Month of March. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

66. City of Edmonton. 1986. Water Treatment Plants Monthly Report - 1986, Month of April. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

67. City of Edmonton. 1986. Water Treatment Plants Monthly Report - 1986, Month of May. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

68. City of Edmonton. 1980. Water Treatment Annual Report 1980. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

69. City of Edmonton. 1981. Water Treatment Annual Report 1981. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

70. City of Edmonton. 1982. Water Treatment Annual Report 1982. Water and Sanitation, Water Treatment Division, City of Edmonton, Alberta.

71. Halina, G. 1983. Treated Water Survey for Calgary, Edmonton, Drumheller, Fort McMurray, Grande Prairie, Lethbridge, Medicine Hat, Red Deer -1983. Municipal Engineering Branch, Alberta Environment, Edmonton.

72. Halina, G. 1984. Treated Water Survey for Calgary, Edmonton, Drumheller, Fort McMurray, Grande Prairie, Lethbridge, Medicine Hat, Red Deer, Olds, Camrose, Wetaskiwin -1984. Municipal Engineering Branch, Alberta Environment, Edmonton.

73. Alberta Environment 1985-86. Treated Water Survey for Edmonton Rossdale and Edmonton E.L. Smith Water Treatment Plants -1985and first quarter 1986. Municipal Engineering Branch, Alberta Environment, Edmonton.

74. Enviro-Test. 1983. Target & Nontarget Analysis of Five Samples from The City of Edmonton Water Treatment Plants - Dec. 22, 1983. Prepared for Edmonton Water & Sanitation, by Enviro - Test Laboratories, Edmonton.

75. Enviro-Test. 1983. Target & Nontarget Analysis of Five Samples from The City of Edmonton Water Treatment Plants - Dec. 19, 1984. Prepared for Edmonton Water & Sanitation, by Enviro - Test Laboratories, Edmonton.

76. Enviro-Test. 1985. Chemical Analysis Report - March 25, 1985. Prepared for the City of Edmonton, Water and Sanitation, by Enviro - Test Laboratories, Edmonton.

77. Enviro-Test. 1985. Chemical Analysis Report - April 1, 1985. Prepared for the City of Edmonton, Water and Sanitation, by Enviro - Test Laboratories, Edmonton.

78. Health and Welfare Canada. 1986. National Survey of Halogenated Phenols in Canadian Drinking Water Supplies. Health and Welfare Canada.

79. A.W.W.A. 1976. Handbook of Taste and Odor Control Experiences in the U.S. and Canada. The American Water Works Association, Denver, Colorado.

80. Walker, G.S., F.P.(T. Lee, and E. Marco Aieta. 1986. Chlorine Dioxide for Taste and Odor Control. *J. Am.Water Works Assoc.* **78**:84-93.

81. Zoeteman, B.C.J., A.J.A. Kraayeveld, G.J. Piet. 1971. Oil Pollution and Drinking Water Odour. *H_2O* . 4:367-371.

82. Lillard, D.A., J.J. Powers, and R.C. Webb. 1975. Aqueous Odor Thresholds of Organic Pollutants in Industrial Effluents. National Environmental Research Center, U.S. E.P.A., Corvallis, Oregon. EPA-660/4-75-002. 20pp.

83. Hawrysh, Z. 1986. Interdepartmental Correspondence Dated March 13, 1986, concerning panel evaluations of Edmonton water. University of Alberta, Dept. of Foods and Nutrition, Faculty of Home Economics, Edmonton.

DRAFT

84. Stevens, A.A. 1982. Reaction Products of Chlorine Dioxide. *Environ. Health Persp.* **46**:101-110.

85. Guadagni, D.G., R.G. Buttery, and S. Okano. 1963. Odour Thresholds of Some Organic Compounds Associated with Food Flavours. *J. Sci. Fd. Agric.* **14**:761.

86. Moore, J.E., L.J. Forrester, and P. Pelosi. 1976. Specific Anosmia to Isobutyraldehyde: The malty primary odor. *Chem. Senses and Flavour* **2**:17-25.

87. Dravnieks, A. Atlas of Odor Character Profiles. American Society of Testing and Materials. ASTM data series **DS 61**.

88. Dowty, B.J., D.R. Carlisle, and J.L. Laseter. 1975. New Orleans Drinking Water Sources Tested by Gas Chromatography - Mass Spectrometry: Occurrence and Origin of Aromatics and Halogenated Aliphatic Hydrocarbons. *Environ. Sci. & Technol.* **9(8)**:762-765.

89. Rice, R.G., and M. Gomez-Taylor. 1986. Oxidation Byproducts from Drinking Water Treatment. Presented at the Second National Conference on Drinking Water, Edmonton, Canada.

90. Rav-Acha, C. 1984. The Reactions of Chlorine Dioxide with Aquatic Organic Materials and Their Health Effects. *Water Res.* **18(11)**:1329-1341.

91. Kennaugh. 1957. Action of Diaphanol on Arthropod Cuticles. *Nature*. **180**:238.

92. Le Cloirec, C., and G. Martin. Evolution of Amino Acids in Water Treatment Plants and the Effect of Chlorination on Amino Acids. (In) Water Chlorination: Chlorine demand reactions: proteins and other organics.

93. Safe Drinking water Committee. 1980. Toxicity of Selected Drinking Water Contaminants. (In) Drinking water and health. **Vol.3** National Academy Press, Washington, D.C., pp67-265.

94. Miltner, R.J. 1977. Measurement of Chlorine Dioxide and Related Products. Water Supply Research Division. Municipal Environmental Research Laboratory, U.S. Environmental Protection Agency, Cincinnati, Ohio. 11pp.

95. U.S. EPA. 1978. Federal Register. U.S. Environmental Protection Agency. Part II. Thursday, February 9, 1978. **43**(28).

96. Lippy, E.C. 1983. Review of Treatment Practices - Rossdale Water Treatment Plant Edmonton, Alberta.

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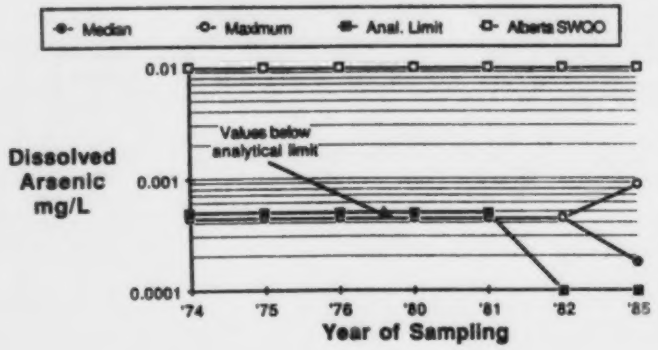
Appendix A

Historical Water Quality in the North Saskatchewan River at Edmonton and Upstream

North Saskatchewan River at Whirlpool Pt.



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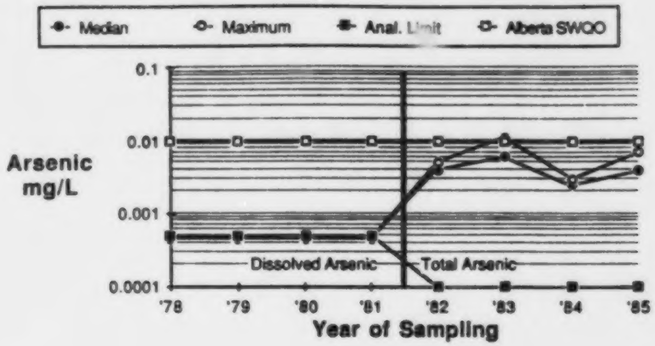

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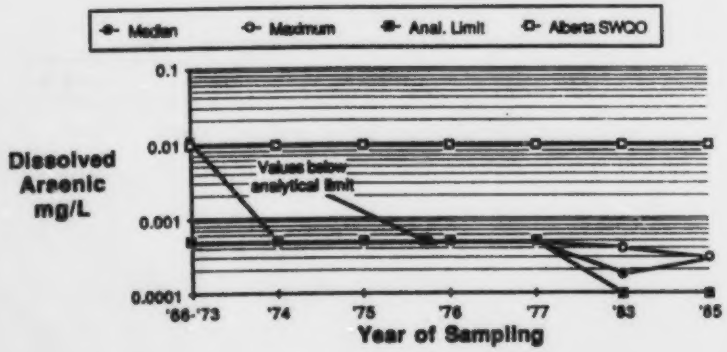

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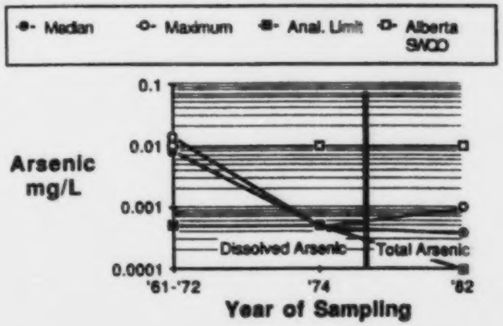

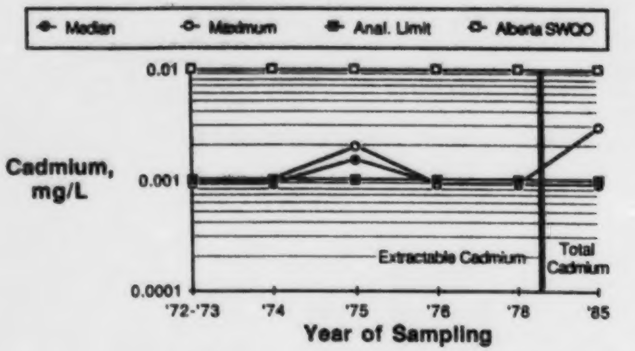

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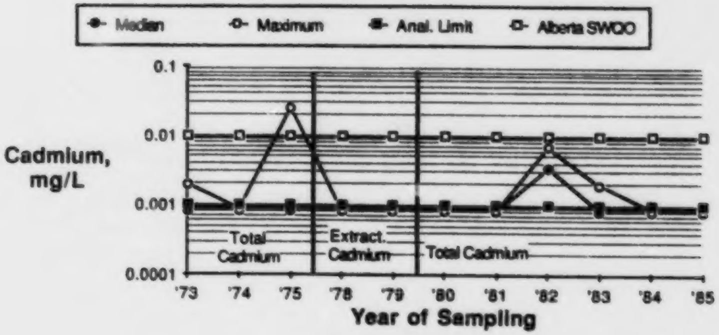

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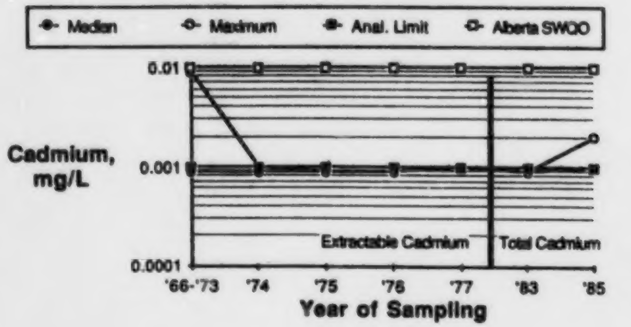

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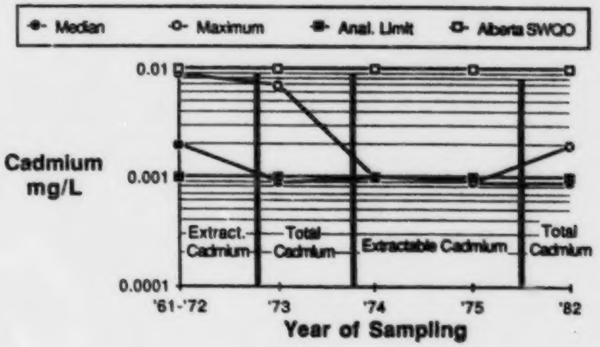

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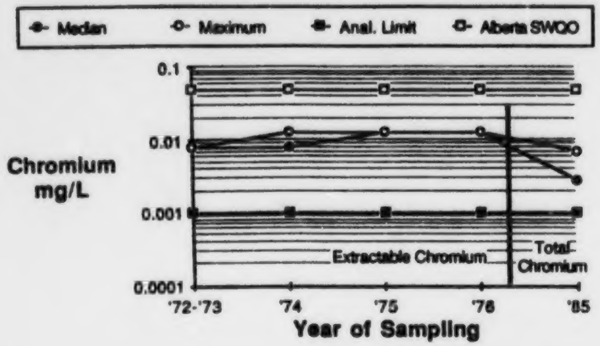

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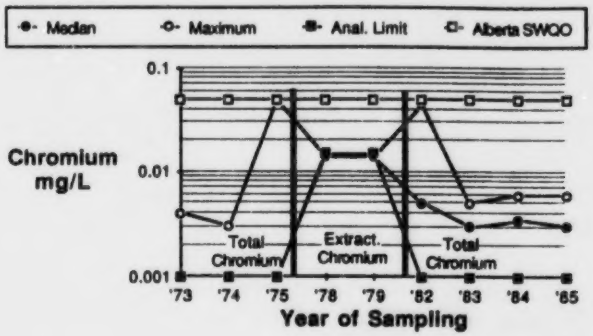

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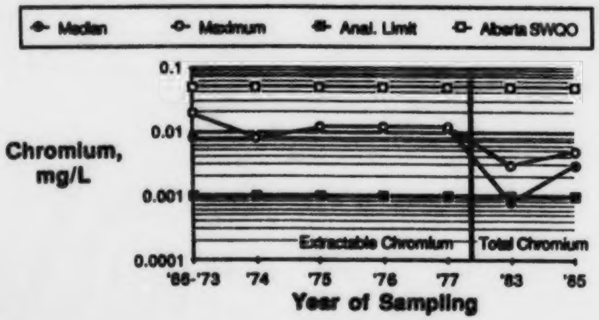

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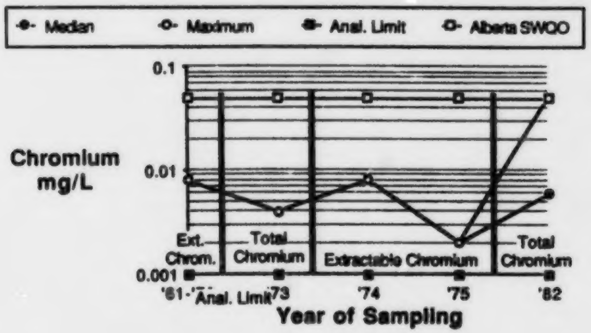

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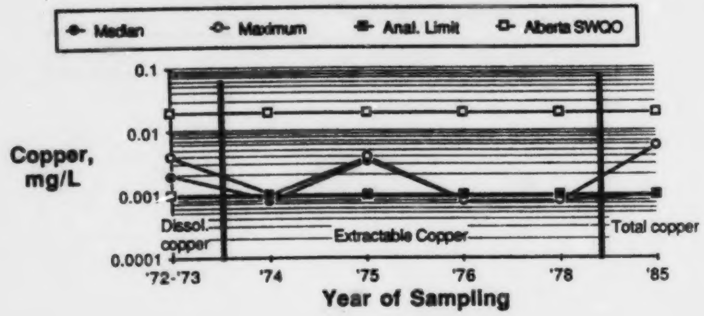

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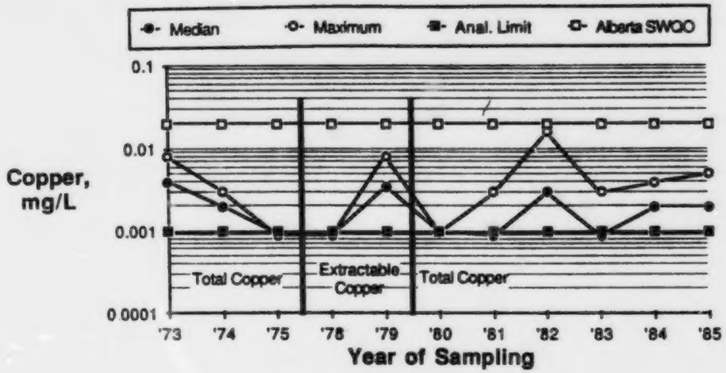

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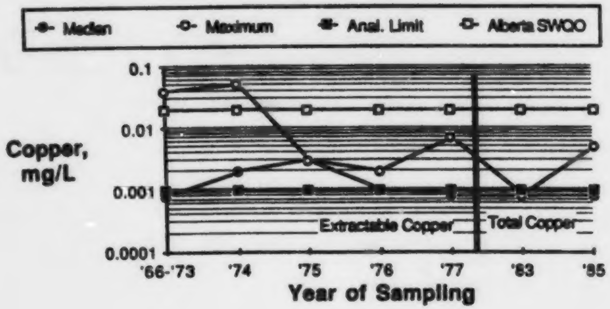

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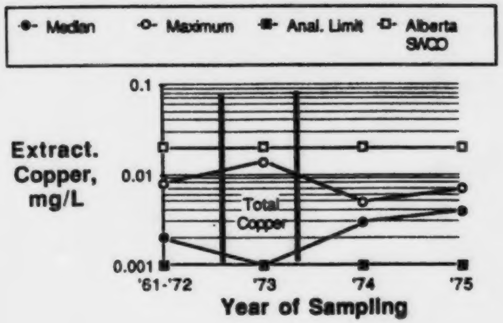

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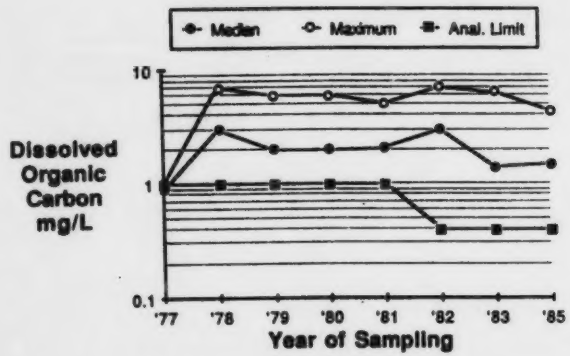

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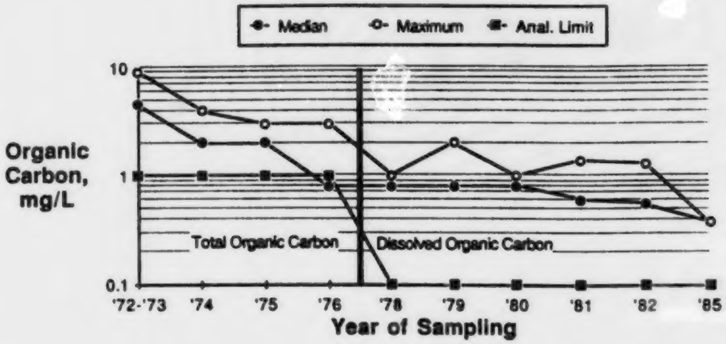

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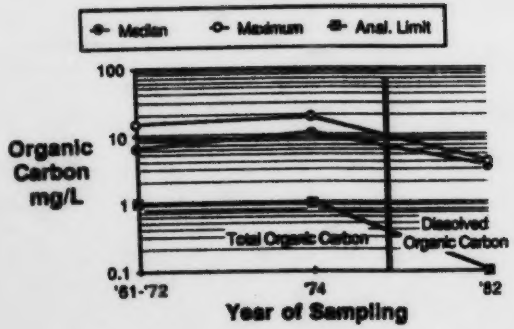

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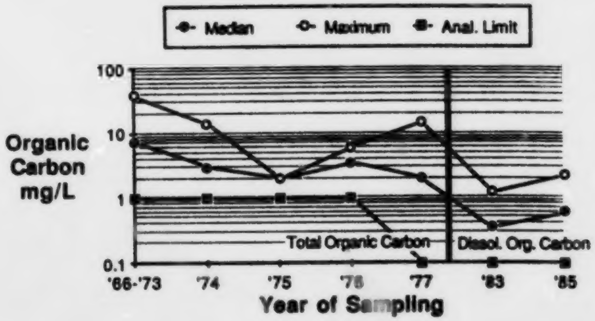

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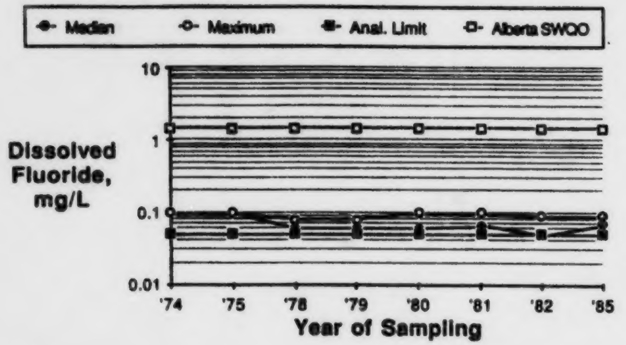

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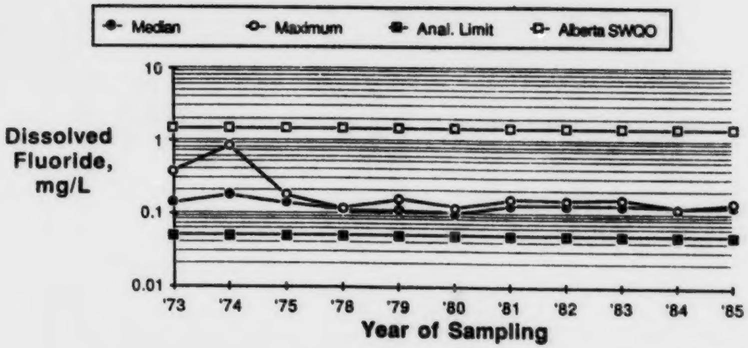

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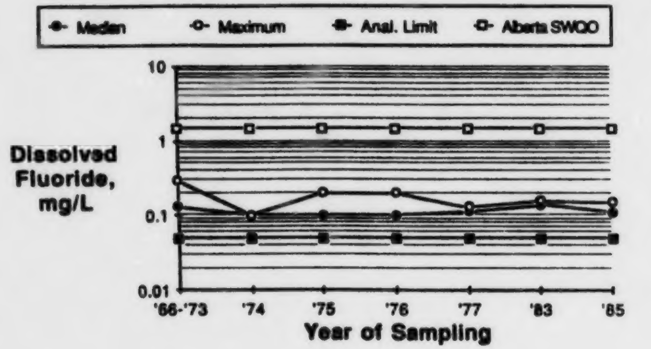

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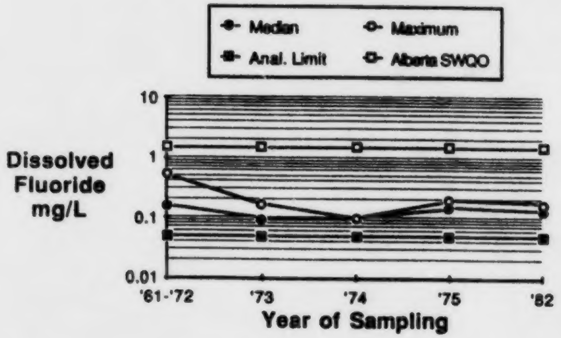

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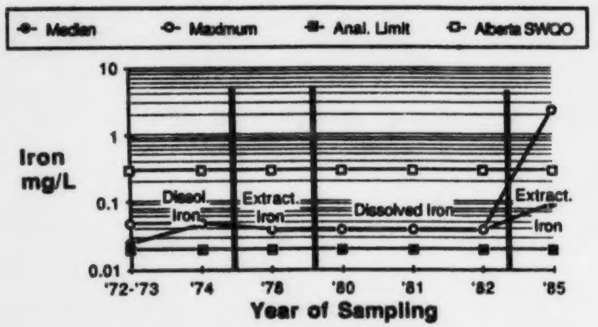

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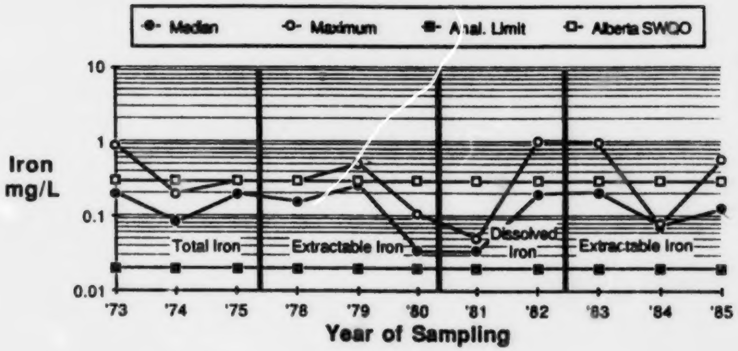

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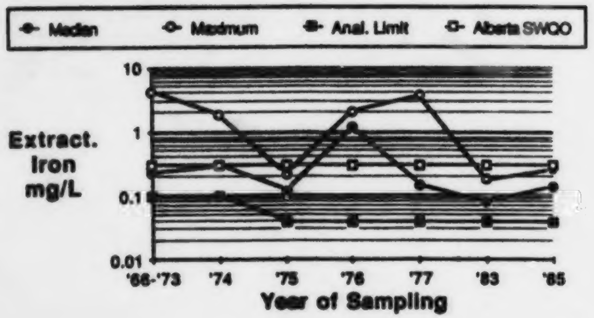

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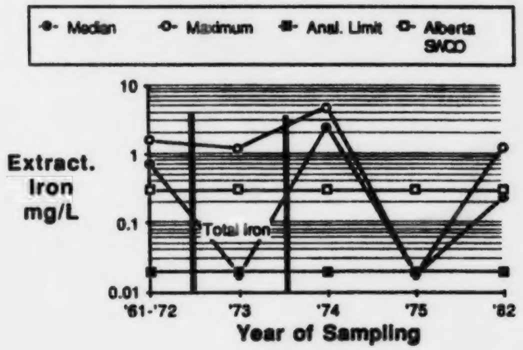

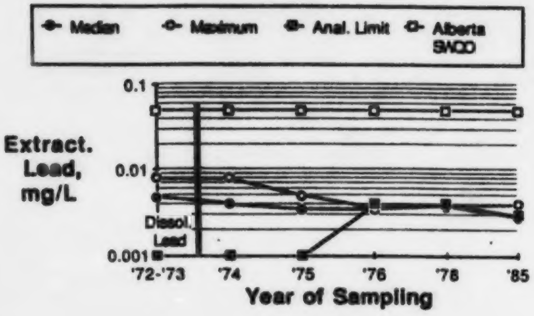
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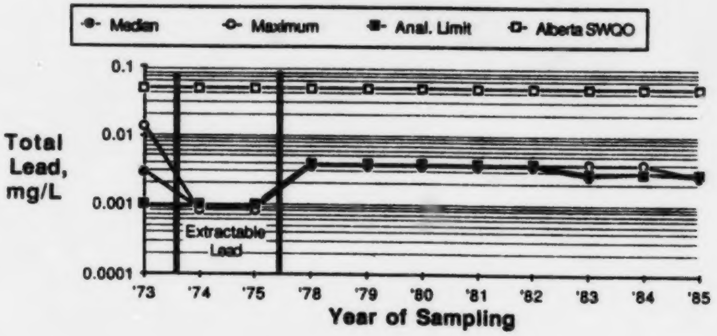
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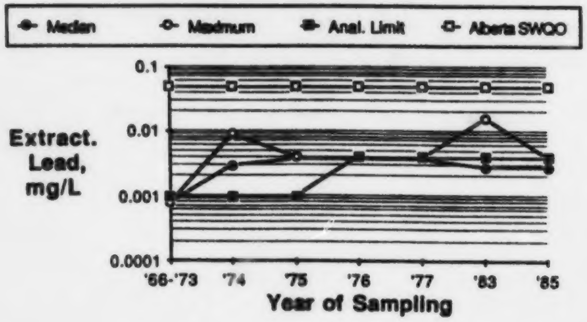
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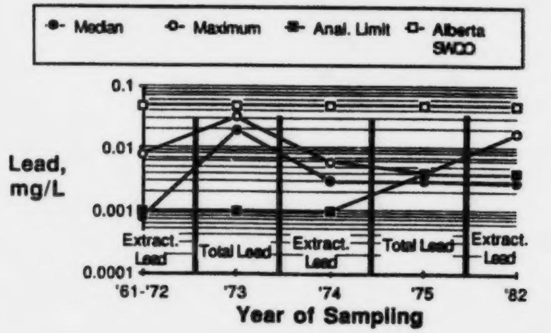
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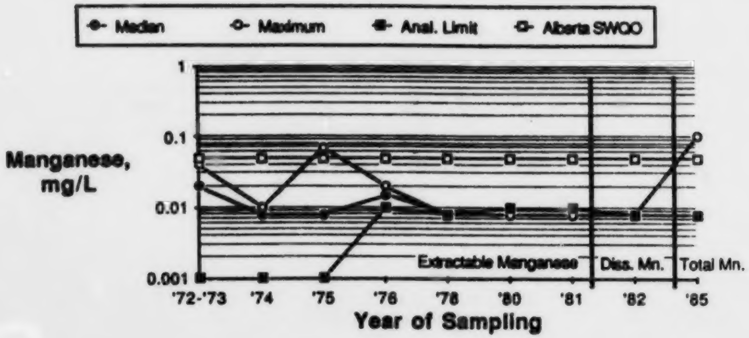

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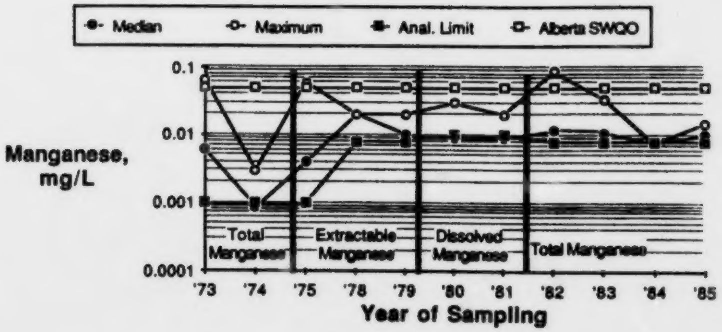

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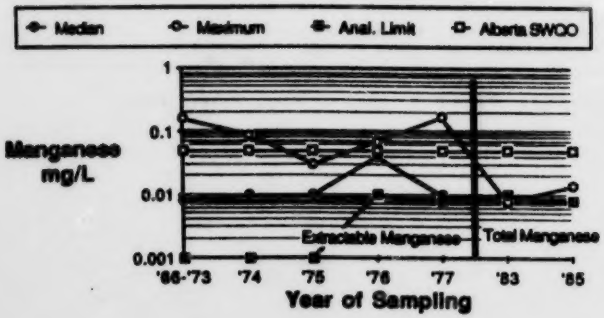

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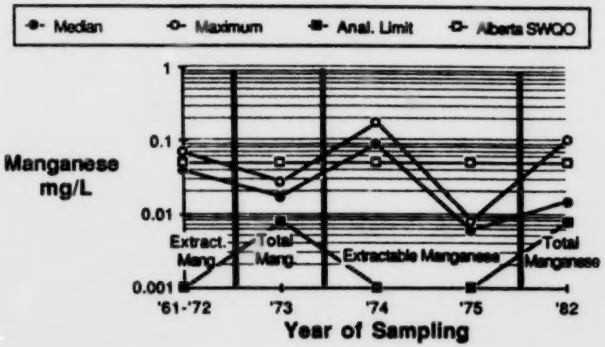

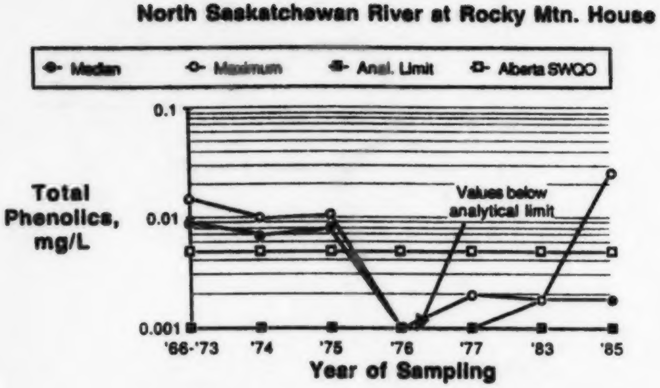
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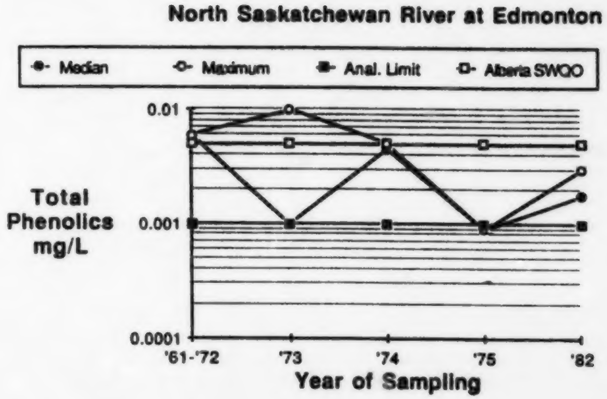
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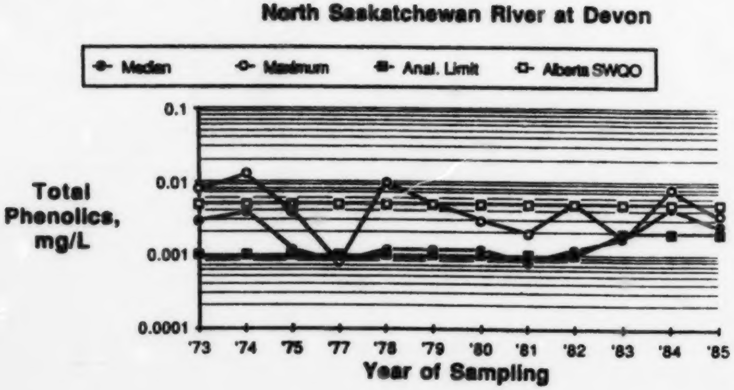
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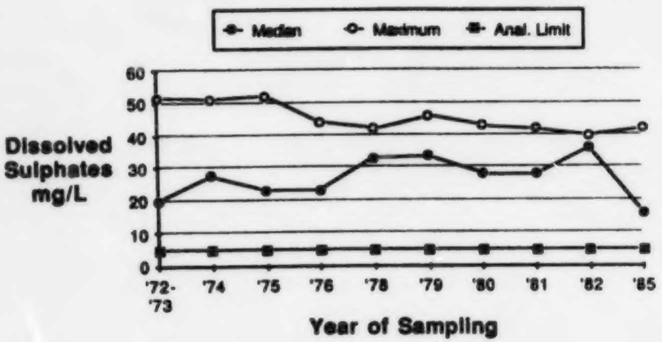
North Saskatchewan River at Whirlpool Pt.



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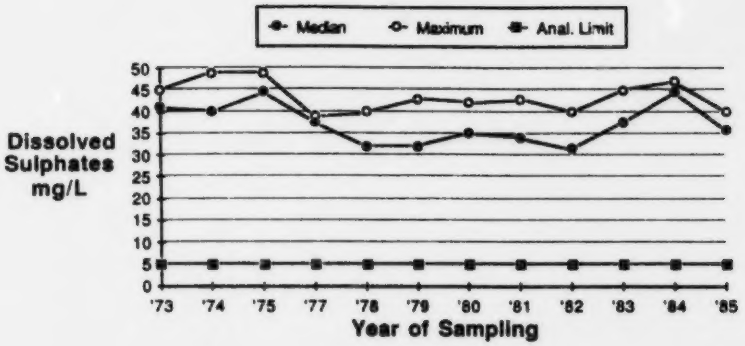

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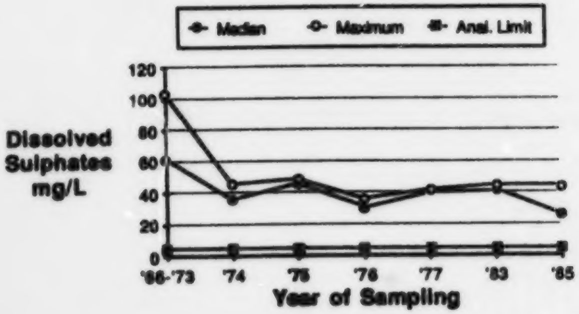

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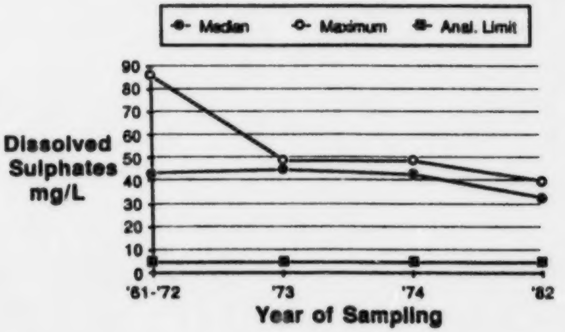

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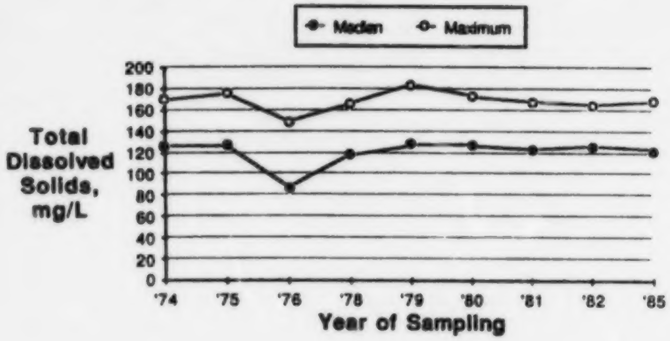

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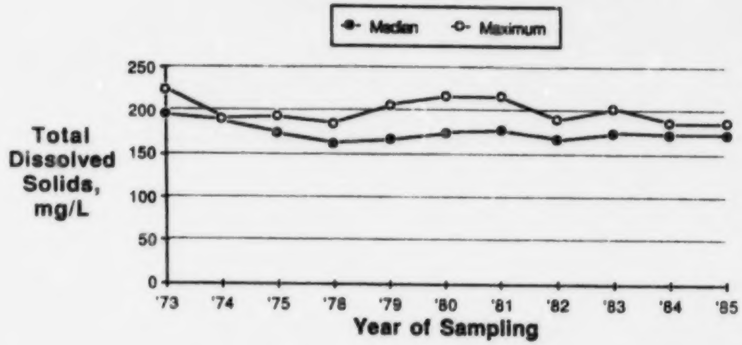

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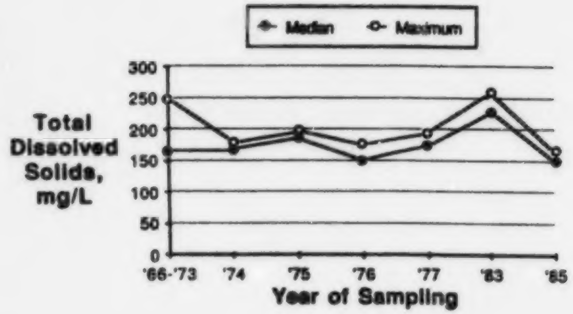

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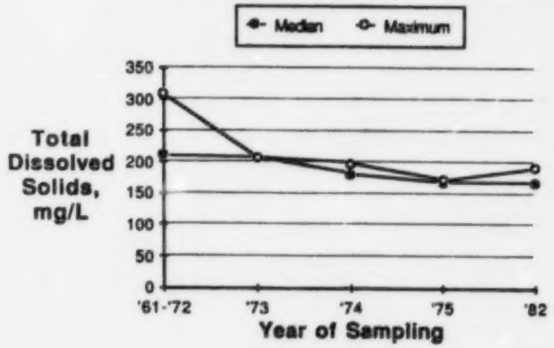

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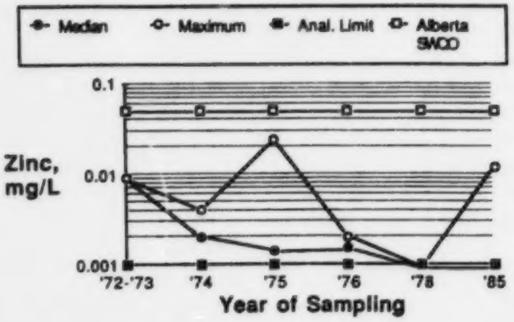

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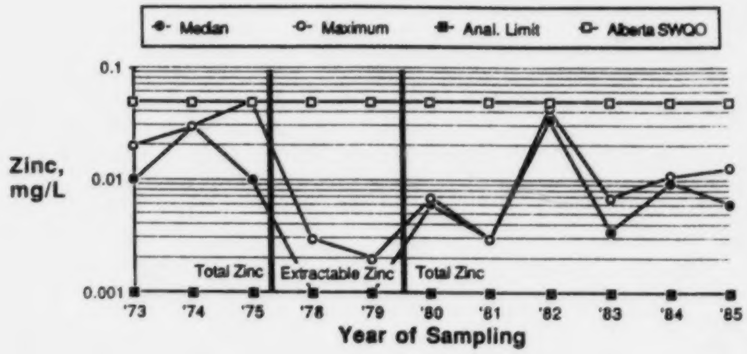

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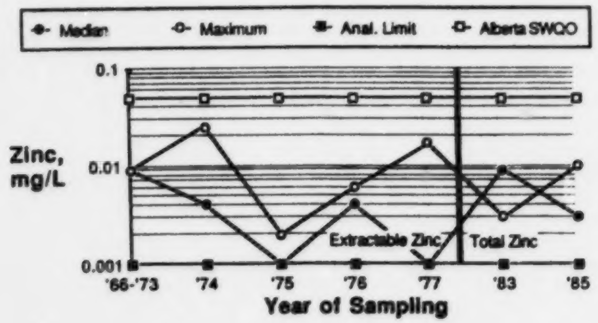

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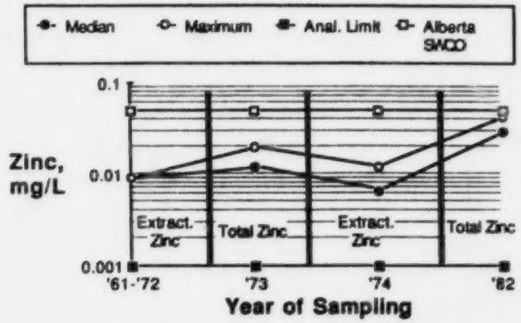


Appendix B

Enviro-Test Laboratories Analytical Procedures for Spring 1986 Monitoring Program

ETL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 - 67th Avenue – Edmonton, Alberta T6E 0P5 Telephone: (403) 434-9509

ANALYSIS OF TARGET PARAMETERS IN EDMONTON RAW AND FINISHED WATER SAMPLES
SPRING RUNOFF 1986

Submitted to:
THE CITY OF EDMONTON
WATER AND SANITATION

April 6/1986

REF #CTYED02.REP
CERTIFIED BY : ____________________
Dennis Erickson, Analytical Chemist

OVERVIEW:

Water samples both raw and finished were analyzed for selected parameters over a 22 day period covering February 25, 1986 to March 18, 1986. The survey was a concentrated effort to determine problem areas during spring run-off.

LIST OF SELECTED PARAMETERS:

1) Extractable Hydrocarbons
2) Purgeable Hydrocarbons
3) Dissolved Organic Carbon
4) Chloride
5) Conductivity
6) Turbidity

METHODOLOGIES:

1. Extractable Hydrocarbons;

A 2 litre sample is transferred to a 2 litre seperatory funnel. The sample is spiked with the following surrogate standards:

1) n-Propylbenzene (50ppb spike)
2) Naphthalene-d_8 (50ppb spike)
3) 4-Bromophenol (90ppb spike)
4) 2-Fluorobiphenyl (50ppb spike)

The surrogate spikes are added to check extraction efficiency in each sample and to establish a solid quality control data base to support the overall procedure.

The 2 litre sample is acidified to pH<2 with the addition of 6N H_2SO_4. The sample is then extracted twice with 125ml of dichloromethane (DCM). The DCM layers are dried through pretreated sodium sulphate (heated in a muffle furnace overnight at 540°C). The DCM extracts are reduced to a small volume via rotary evaporation and the extract is quantitatively transferred to a 3 1/2ml vial using hexane for the final rinsing (Note the samples were never reduced to complete dryness as this would reduce the concentration of the volatile components). The final extracts are reduced to exactly 2ml using a gentle stream of nitrogen. The final concentrates are injected on a gas chromatograph/flame ionization (GC/FID) system and semi-quantitated against a calibrated diesel standard. Calibrated surrogate standards are also compared by external calculation to determine percentage recoveries. All results are reported in parts per billion (ppb). The final value represents a semi-quantitative amount of total extractable hydrocarbons.

The analysis is completed using a DB-1 capillary column, 30m X .32mm I.D. with a 1um film thickness. The oven is programmed from 40°C for 4 minutes to 300°C at 10°C/minute with a 10 minute hold time. The injection port and the detector were set at 250°C.

Samples were extracted immediately upon receipt and the extracts were analyzed as soon as possible. All extracts are being stored in a freezer and can be analyzed in more detail if desired.

The detection limit for a 2ml final volume was set at 10ppb however this can be reduced by concentrating the final extract to a smaller volume if desired.

2. Purgeable Hydrocarbons;

A 1.6 litre sample is purged with nitrogen at 500ml/minute for 30 minutes. The volatile organic compounds are adsorbed into standard (150mg) activated charcoal tubes (Supelco, cat #2-0267). The sparging and collection apparatus consists of a 2 litre erlenmeyer flask equipped with a rubber stopper. A gas dispersion tube of medium-porosity fritted glass and an adsorption train are inserted (gas-tight) into the stopper. The adsorption train consists of:

a) Drying tube - 13cm X 6mm i.d. glass tubing filled with 10/20 mesh Drierite with glass wool plugs at both ends.

b) An activated coconut charcoal tube (MSA, 150mg size) connected to the drying tube with a short piece of tygon tubing.

A sampling train of six flasks were purged at one time thus allowing the completion of up to 22 samples per day.

After completion of sparging, the activated carbon cartridge is opened and its contents (front and back) are transferred to a 2ml autosampler vial. One ml of carbon disulphide (CS_2) is added, the vial is capped and shaken for 1/2 hour.

A surrogate standard, n-Propylbenzene (50ppb) was added to each sample before purging to ensure method efficiency.

The extracts are analyzed by GC/FID using the column outlined in the extractable hydrocarbons.

The oven in programmed from 40°C for 8 minutes to 150°C at 10°/ minute with a 10 minute hold time. The injection port and the detector were set at 250°C.

Samples were purged immediately upon receipt and the extracts were analyzed as soon as possible. All extracted are being stored in a freezer and can be analyzed in more detail if desired.

The detection limit was set at 5ppb based on the purging efficiency and response of n-Propylbenzene.

See Appendix 3 for a copy of a paper describing the method in more detail.

The final calculations are completed by using n-Propylbenzene as standard and comparing the total integrated areas of the samples with that of n-Propylbenzene.

3. Dissolved Organic Carbon;

The analysis is performed using an Autoanalyzer system. The sample is acidified with sulphuric acid then an autosampler introduces the sample into the system. The sample is parged with nitrogen gas to remove any CO_2 from inorganic carbon. Additional acid and potassium persulphate are added. The solution is then passed to an ultraviolet digestor exposed to U.V. light where the organic carbon is converted to CO_2. The sample is then passed to a dialyzer where CO_2 is dialyzed across a gas permeable membrane into a pH 9.30 phenolphthalein solution and the color loss is measured at 500nm and compared to phthalate carbon standards. The range is 0.5 to 20mg/L.

4. Chloride;

Chloride is measured in an Autoanalyzer system. The chloride introduced by the autosampler is mixed with mercuric thiocyanate. The thiocyanate ion displaced from the mercuric thiocyanate by the chloride ion is reacted with ferric ion to form a red coloured complex, ferric thiocyanate. The concentration of the coloured complex, which is proportional to the original chloride concentration, is measured colorimeterically at 480nm. The range is .1 to 5.0mg/L.

5. Conductivity;

The conductivity is measured directly using a platinum electrode cell. The resistance measured across the cell electrodes is read directly in uS/cm using a Radiometer CDM 83 conductivity meter. The meter is calibrated using standard potassium chloride solutions of .01 M and .05 M.

6. Turbidity;

The analysis is performed using a direct reading nephelometer. The sample is put into a glass cuvette. Light passed through the sample is scattered by the suspended particles. The intensity of the light, which is reflected at 90° to the light beam is measured, and is directly proportional to the turbidity of the sample. The turbidimeter is calibrated using a formazin solution. The range is 0.1 to 30.0 NTU.

QUALITY CONTROL:

1. Organic Analysis;

Some concern was expressed as to whether or not the two methods would cover the complete boiling range of hydrocarbons, that is purgeable volatiles to extractable semi-volatile components. As a results n-Propylbenzene was chosen as a suitable surrogate which falls in the range of both a purgeable (bp 160°C) and an extractable. Data for the recovery of this compound using both methods is detailed in Appendix 1.

All of the purgeable samples were spiked with n-Propylbenzene. The majority of the extractable samples were spiked with all four surrogates. In reviewing the data with the project manager (Dr. Steve Hrudy) and his colleagues (Dr. R. Coutts and Dr. Kratochvil) it was decided that only 10% of the samples be spiked for the last 8 days of the program. This decision was made due to the overall consistency of the recoveries and the suspicion that these four compounds may be masking smaller concentrations of unknowns which could be eluting at the same retention times.

The accuracy of the method is completely detailed in Appendix 1. Appendix 1 is a presentation of the surrogate spike accuracy at the beginning of the program (February 25 & 26) and towards the end of the program (March 4 & 5 - Purgeables, March 7 & 8 - Extractables). The accuracy is demonstrated by plotting the data in a format standard to the EPA ("Handbook for Analytical Quality Control in Water and Wastewater Laboratories, March 1979, EPA 600/4-79-019), see Appendix 2 for excerpts from the reference handbook. As can be seen from the charts the methods were in control during these time periods. Naphthalene-d_8 did drop in extraction efficiency from 79% to 48.6%

however the recoveries were consistent during the two time periods. The other surrogate recoveries remained relatively the same through out the course of the study.

The other aspects of the quality control were 10% duplicates and a blank run for each day of extraction and purging. The duplicate results are also presented in Appendix 1, Table 1. The duplicate results on storm sewer samples were not consistent due to the problem of spilting a sample which had a visible oil content.

All blank values (FID area counts) were subtracted from the sample area counts before final calculation.

CS_2 reagent blanks were also analyzed to ensure the absence of benzene and other aromatics which have been reported as being possible contaminants.

2. Inorganic Analysis:

Each day the instruments were calibrated prior to running the City of Edmonton samples with in house prepared standards. With each set of samples one to two duplicates were analyzed to measure precision. An EPA standard was also run with each set for all 4 parameters. Spike recoveries were also run for the organic carbon and chloride. The results of the precision and accuracy data were summarized statistically as follows:

a. Dissolved Organic Carbon;

Accuracy - Spike Average Recovery = 99.1%
Standard Deviation = 6.88%
n = 22

EPA Standard Design 6.5mg/L

Range at 95% confidence 4.4 to 8.1mg/L

Analyzed at average = 6.22mg/L

Standard Deviation = 0.29mg/L

for n = 22

Precision - Duplicates average difference;

= 0.39

Standard Deviation = 0.91

for n = 38

b. Chloride;

Accuracy - Spike average recovery = 98.2%

Standard Deviation = 4.83%

for n = 10

EPA Standard Design 17.8mg/L

Range at 95% confidence 16.1 to 19.9mg/L

Analyzed to average = 17.6mg/L

Standard Deviation = 0.64

for n = 10

Precision - Duplicates average difference;

= .14

Standard Deviation = .17

for n = 22

c. Conductivity (EC);

Accuracy EPA Standard Design 108uS/cm
Range at 95% confidence 95.2 to 119mg/L
Analyzed at average = 107.6
Standard Deviation = 0.64
Precision - Duplicates average difference;
= 3.67
Standard Deviation = 5.5
for n = 18
Range of analysis 200 to 1150uS/cm

d. Turbidity;

Accuracy EPA Standard Design = 4.4
Range at 95% confidence 3.77 to 5.01 mg/L
Analyzed at average = 4.8
Standard Deviation = .32
for n = 9

Precision - Duplicates average difference;
= .28
Standard Deviation = .19
for n = 12

Appendix C

Preliminary Characterization of Humic Materials from the North Saskatchewan River - 1986

The water sample (100 L) was filtered through Millipore filters (0.45 um) to remove the suspended material. The filtrate was thenfractionated into four samples by filtering through the following Millipore membranes : 100,000 Nominal Molecular Weight (NMW), 30,000 NMW, 10,000 NMW and 1,000 NMW. The retentate from each filtration (300 mL) were freeze dried and their spectra obtained at ARC Analytical Chemistry Labs. The samples were named SH100 (NMW > 100,000), SH30 (30,000 < NMW < 100,000), SH10 (10,000 < NMW < 30,000), SH1 (1,000 < NMW <10,000). UV-VIS, IR, and 13C liquid NMR spectra of the four samples are presented in the appendix.

The sample weights recovered are presented in Table 1, it shows that about 500 mg were recovered from the water sample.

Table 1. Weights (mg) of samples recovered from water through filtration.

Sample	SH1	SH10	SH30	SH100
NML	1,000-10,000	10,000-30,000	30,000-100,000	>100,000
UV-VIS	5	5	5	5
IR	2	2	2	2
NMR	218	65	66	53
Left	46	0	0	0
Total	271	72	73	60

The UV-VIS spectra of the four samples is similar to the spectra of humic materials extracted from various sources (waters, soils). The IR spectra of the samples shows the presence of some inorganic impurities, especially those of calcium carbonate and sulphate and Si, other features are similar to those obtained for other humic materials. The 13C NMR spectra showed the presence of carbohydrate materials (50 - 110 ppm range) in the SH1, SH10, SH30, and to much lesser degree in the SH100 sample. Other features included the strong presence of caboxyl carbons (170 ppm) in the SH10, SH30, and SH100 samples and its absence in the SH1 sample.

Examination of samples dissolved in 0.05 N $NaHCO_3$ showed the presence of several elements that are precipitated with the humic material or complexed with it (Table 2). The presence of these elements indicates the importance of humic materials in the transport of various elements (e.g heavy metals) when complexed.

Table 2. The elements present in the dissolved humic materials as detected with Inductively Coupled Plasma Atomic Emission Spectroscopy.

Element	SH1	SH10	SH30	SH100
Ca	***	**	**	**
Mg	***	**	**	**
Si	**	**	***	***
K	*	*	*	*
S	***	**	**	**
B	**	b	b	b
Sr	**	**	**	**
Zn	***	***	***	***
Fe	***	b	b	b

*,**,*** indicated that the element is somewhat higher (< 2X), higher (< 3X), and much higher (> 3X) than background.

b indicates that the elements is not higher than background.

INDUSTRIAL TECHNOLOGIES
ANALYTICAL CHEMISTRY

UV-Vis

NAME : S. Abboud

SAMPLE : SH1

LAB. # : 16-86 UV-Vis

LAMBDA : 220 to 820

A or %T : Absorbance

PATHLENGTH : 1.0 cm

REFERENCE : 0.05N NaHCO3

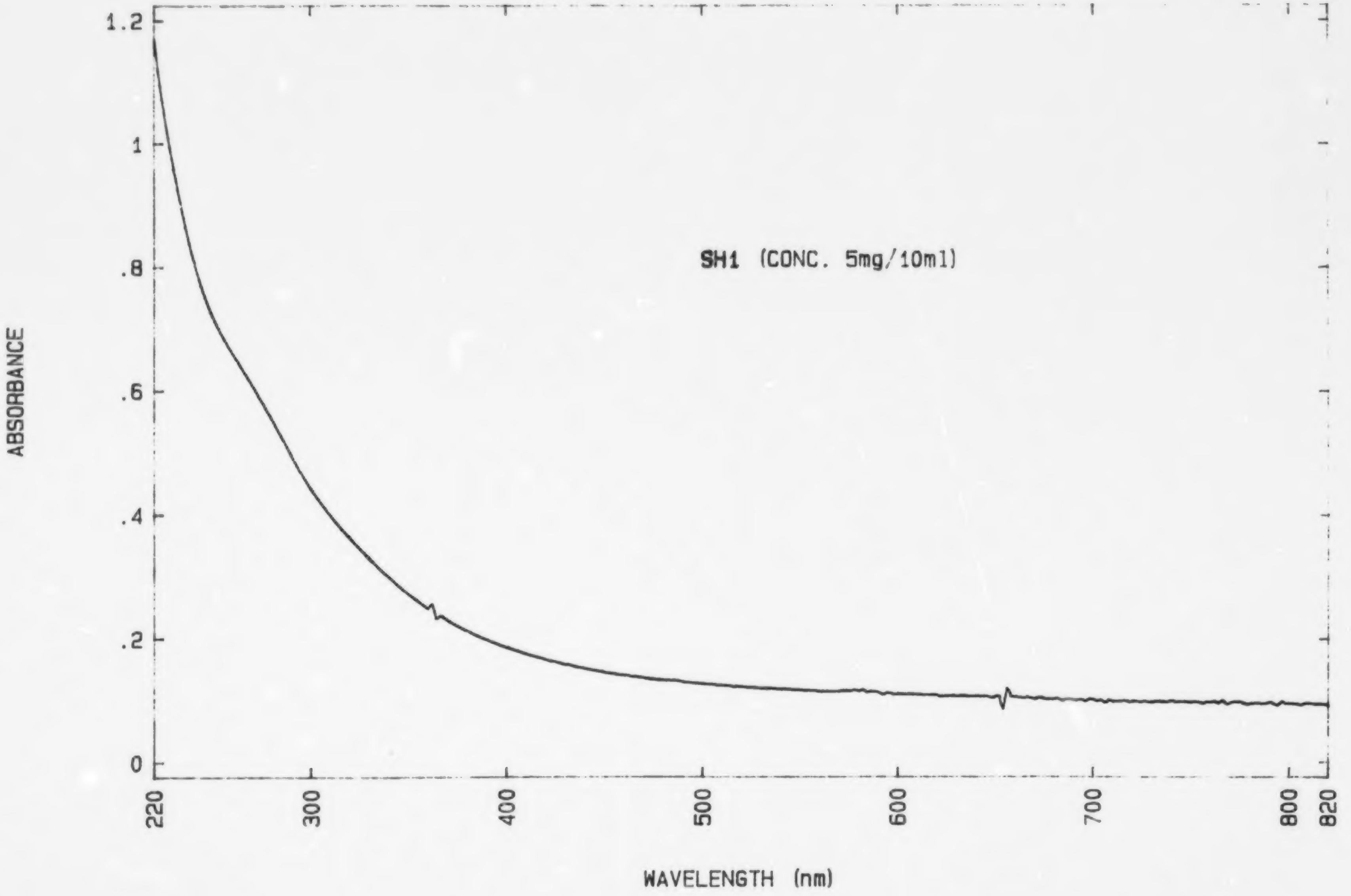
CONCENTRATION : 5mg/10ml

DILUTION FACTOR : N.A

DISK.FILE : ABBOUD.FILE51

DATE : June 4/1986

OPERATOR : C.G



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INDUSTRIAL TECHNOLOGIES
ANALYTICAL CHEMISTRY

UV-Vis

NAME : S. Abboud

SAMPLE : SH10

LAB. # : 19-86 UV-Vis

LAMBDA : 220 to 820

A or %T : Absorbance

PATHLENGTH : 1.0 cm

REFERENCE : 0.05N NaHCO3

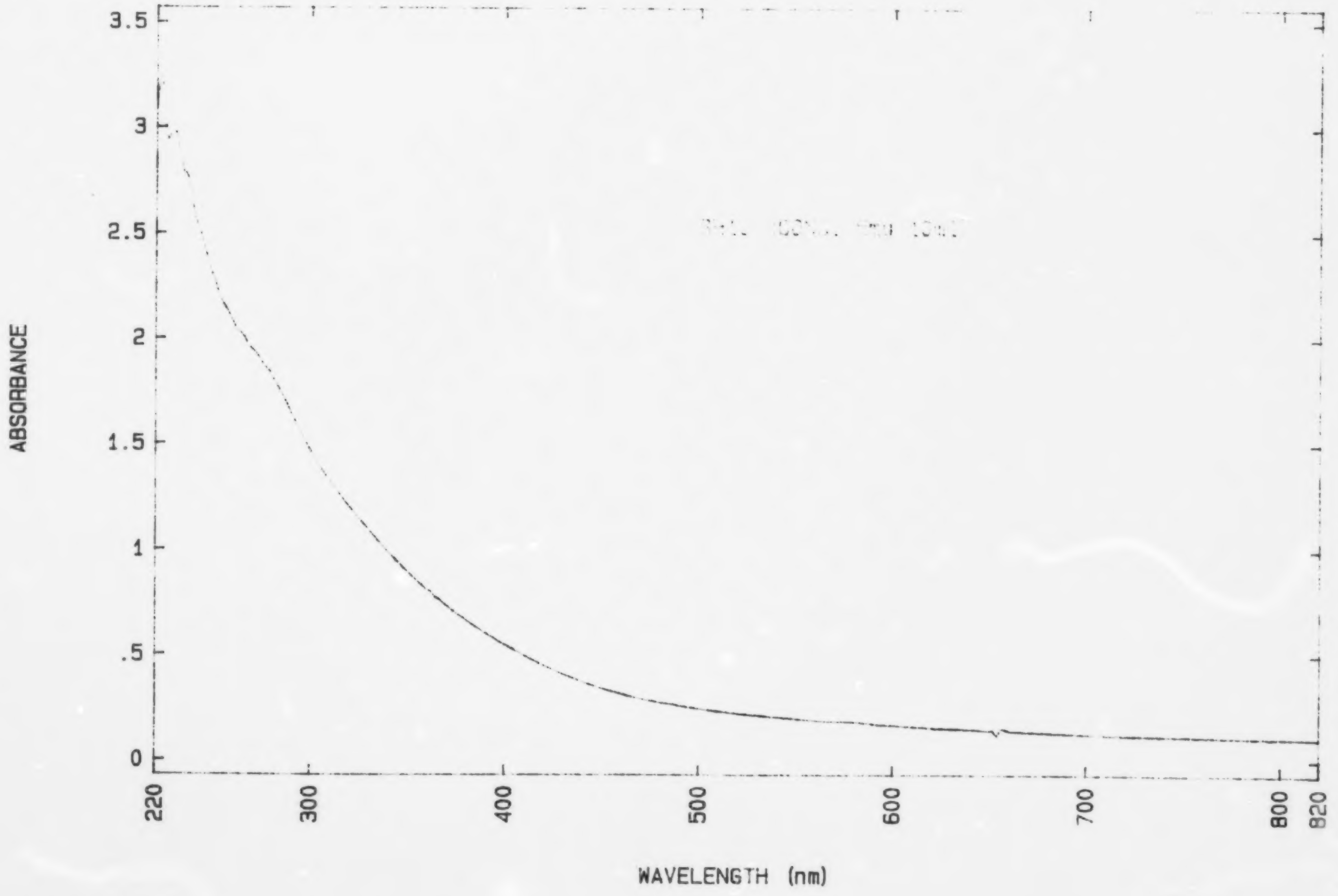
CONCENTRATION : 5mg/10ml

DILUTION FACTOR : N.A.

DISK.FILE : ABBOUD.FILE54

DATE : June 4/1986

OPERATOR : C.G.



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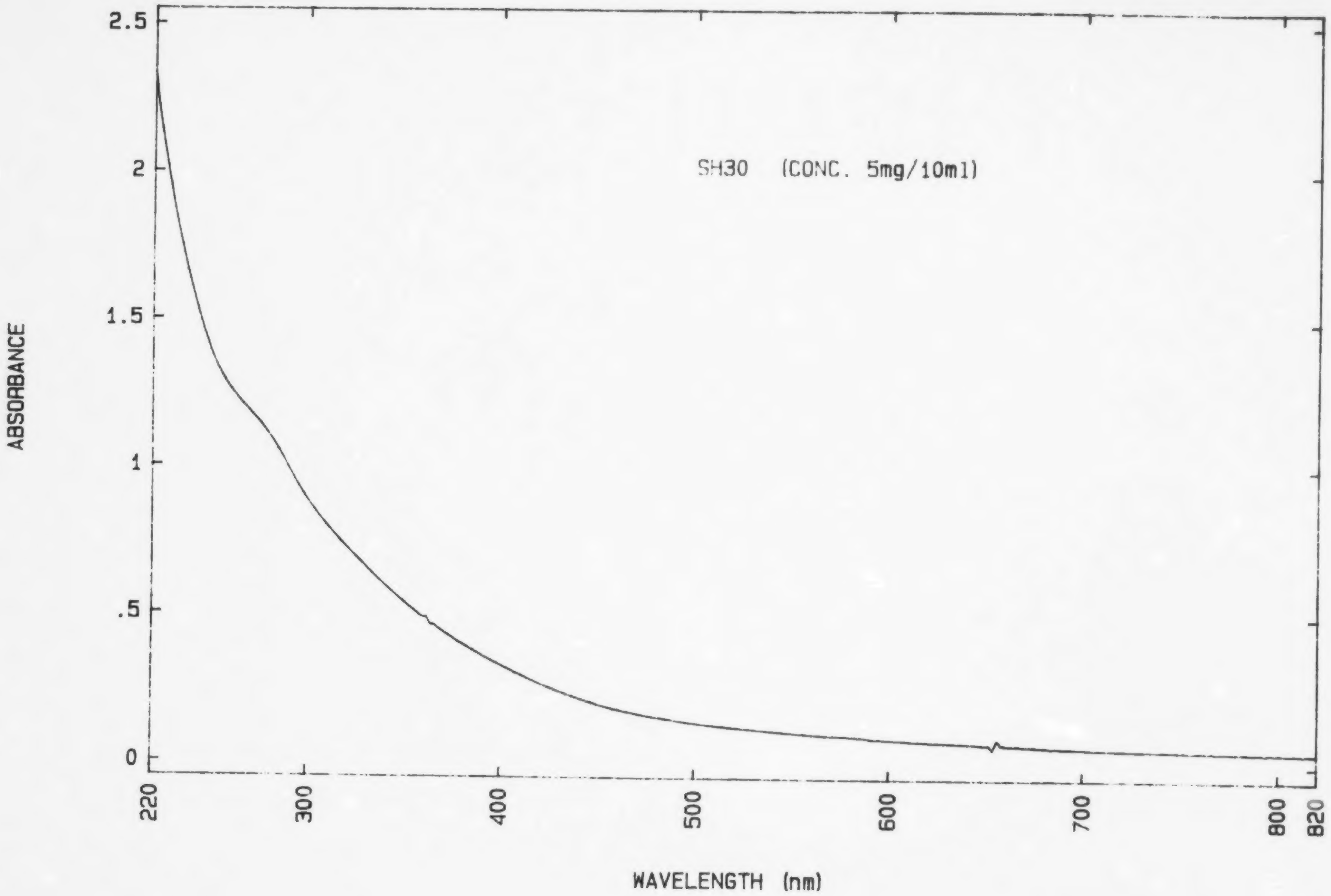
INDUSTRIAL TECHNOLOGIES
ANALYTICAL CHEMISTRY

UV-Vis

NAME	:	S. Abboud
SAMPLE	:	SH30
LAB. #	:	18-86 UV-Vis
LAMBDA	:	220 to 820
A or %T	:	Absorbance
PATHLENGTH	:	1.0 cm
REFERENCE	:	0.05N NaHCO3
CONCENTRATION	:	5mg/10ML
DILUTION FACTOR	:	N.A.
DISK.FILE	:	ABBOUD.FILE53
DATE	:	June 4/1986
OPERATOR	:	C.G



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INDUSTRIAL TECHNOLOGIES - ANALYTICAL CHEMISTRY

INDUSTRIAL TECHNOLOGIES
ANALYTICAL CHEMISTRY

UV-Vis

NAME : S.Abboud

SAMPLE : SH100

LAB. # : 17-86 UV-Vis

LAMBDA : 220 to 820

A or %T : Absorbance

PATHLENGTH : 1.0 cm

REFERENCE : 0.5N NaHCO3

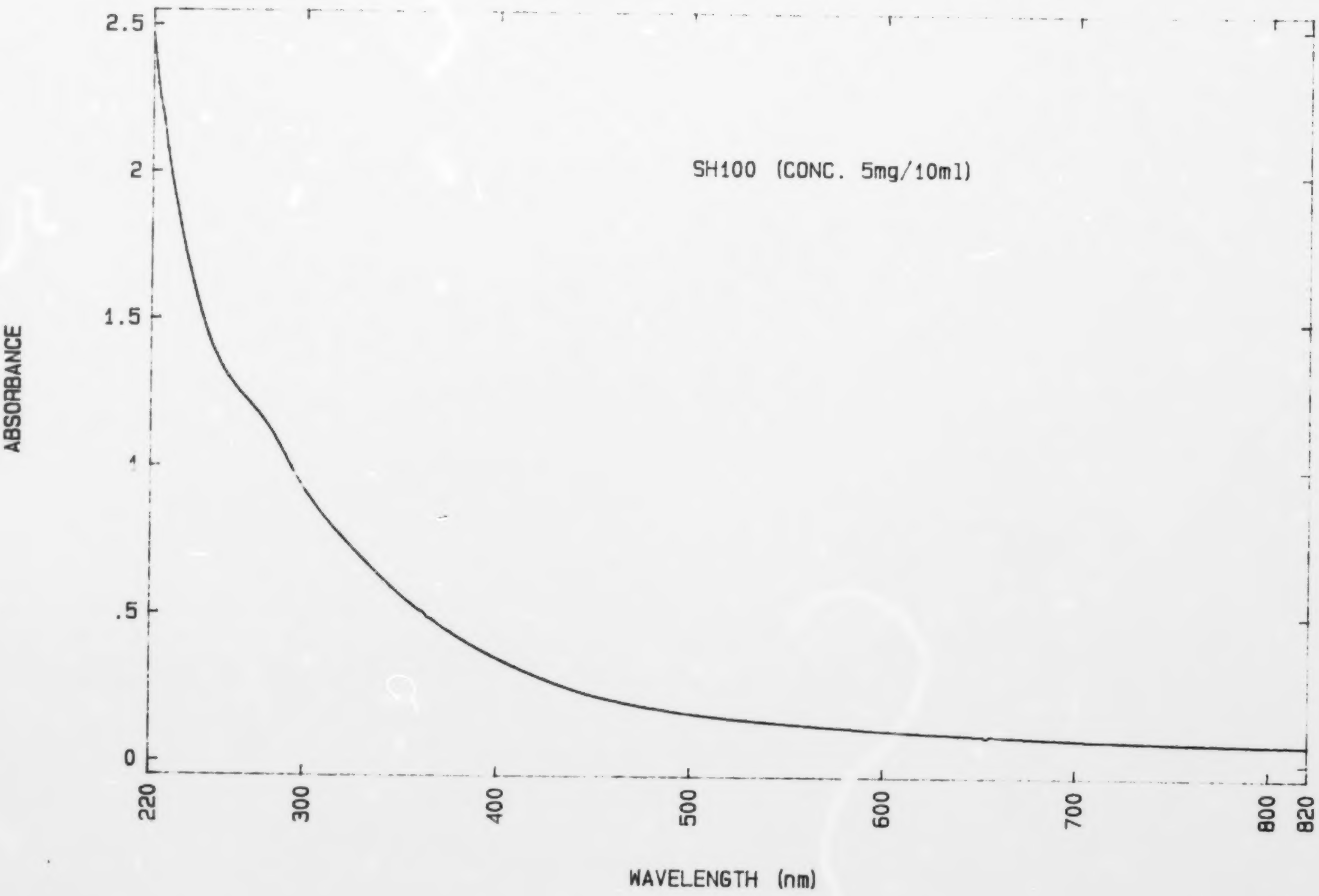
CONCENTRATION : 5mg/10ml

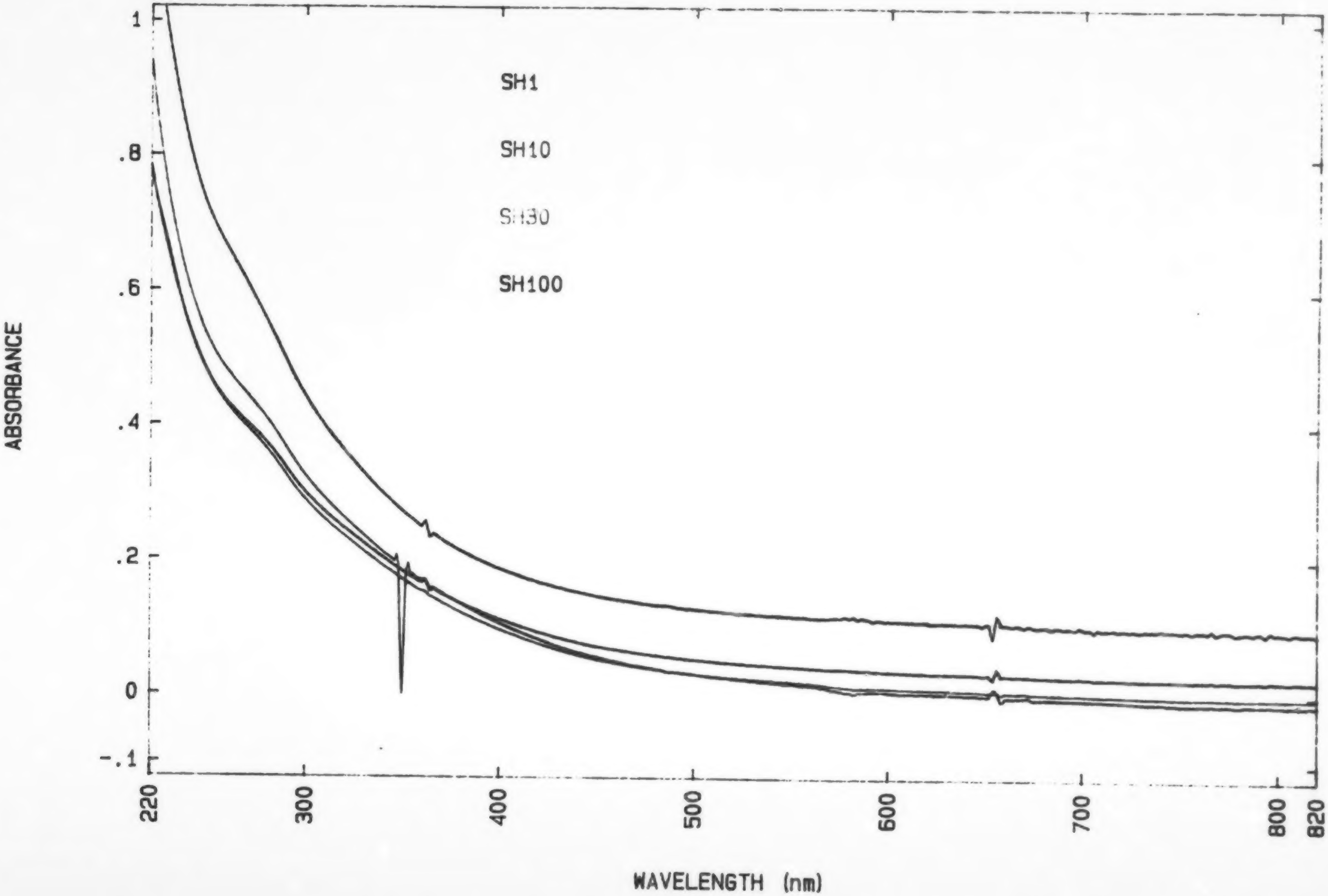
DILUTION FACTOR : N.A.

DISK.FILE : ABBOUD.FILE52

DATE : June 4/1986

OPERATOR : C.G.


SH100 (CONC. 5mg/10ml)
ABSORBANCE
2.5
2
1.5
1
.5
0
220
300
400
500
600
700
800
820
WAVELENGTH (nm)



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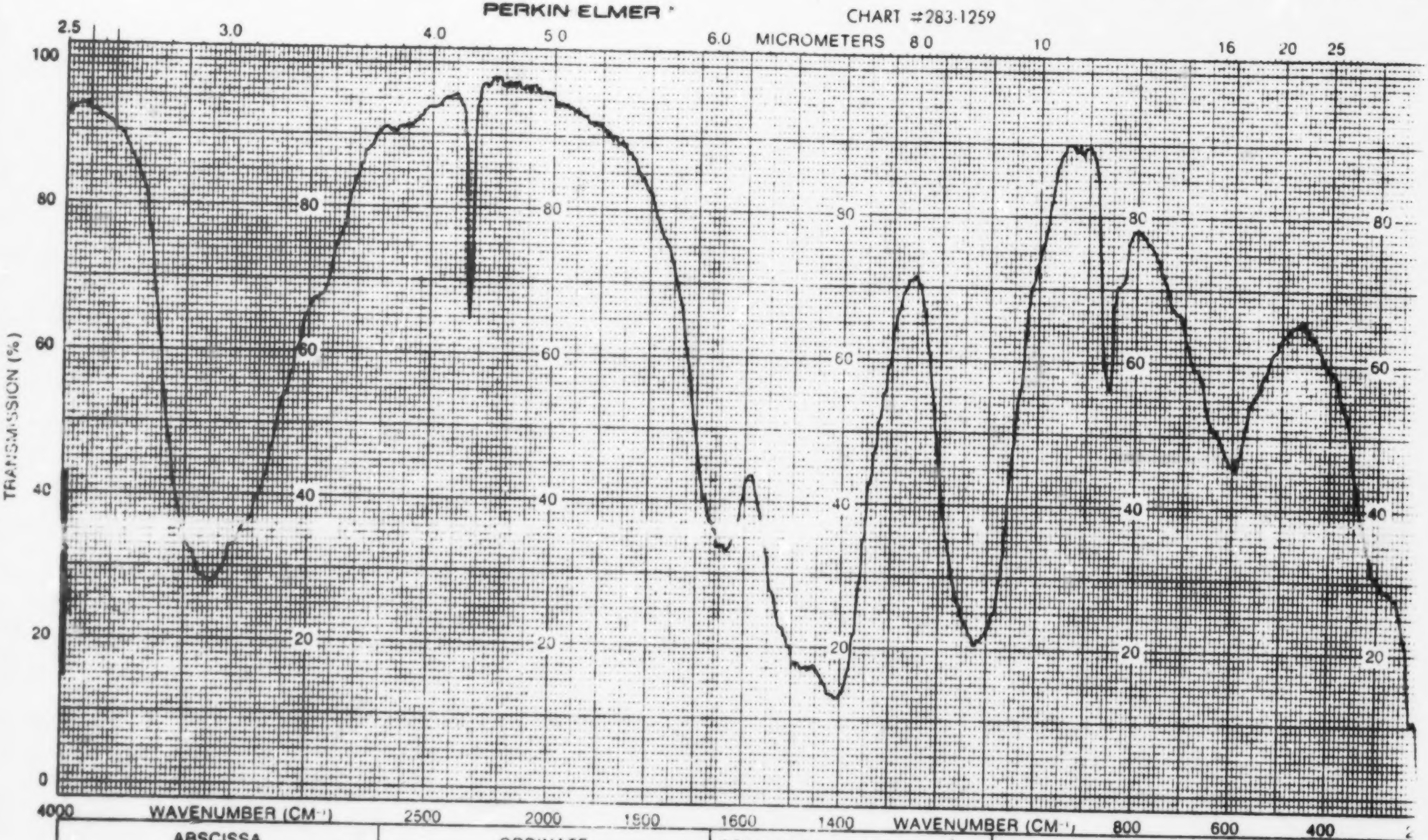

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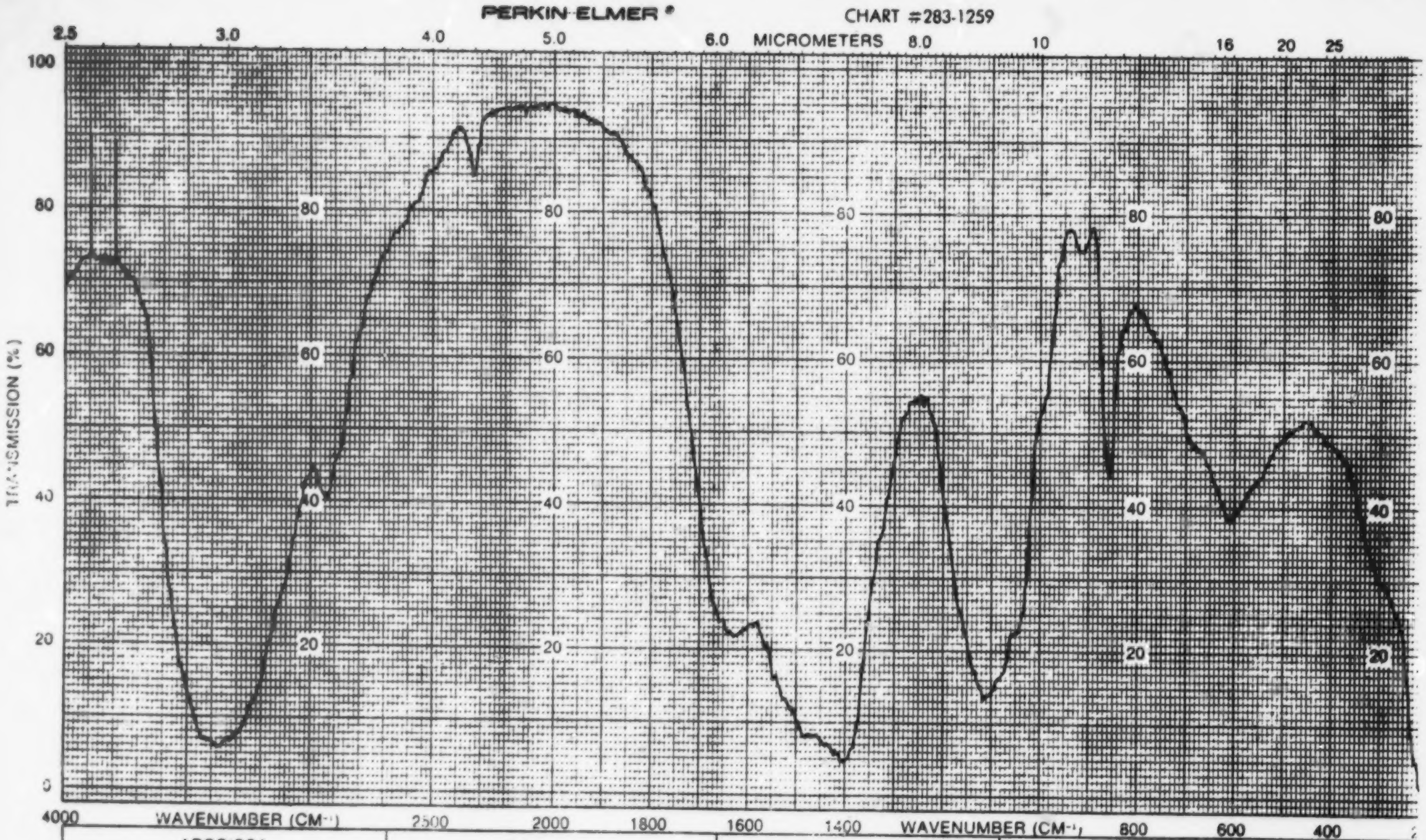
EXPANSION 1
SUPPRESSION
ORDINATE
EXPANSION 1
%T ✓ ABS
SCAN TIME 12 min
RESPONSE 1
SLIT PROGRAM N
REP. SCAN
TIME DRIVE
OPERATOR S.A.
SINGLE BEAM
PRE SAMPLE CHOP
DATE June 4/8
SAMPLE SHI
REMARKS 0.5% KBr
SOLVENT
CONCENTRATION
CELL PATH
ORIGIN Salim Abboud
REFERENCE 163-861R

PERKIN-ELMER® CHART #283-1259

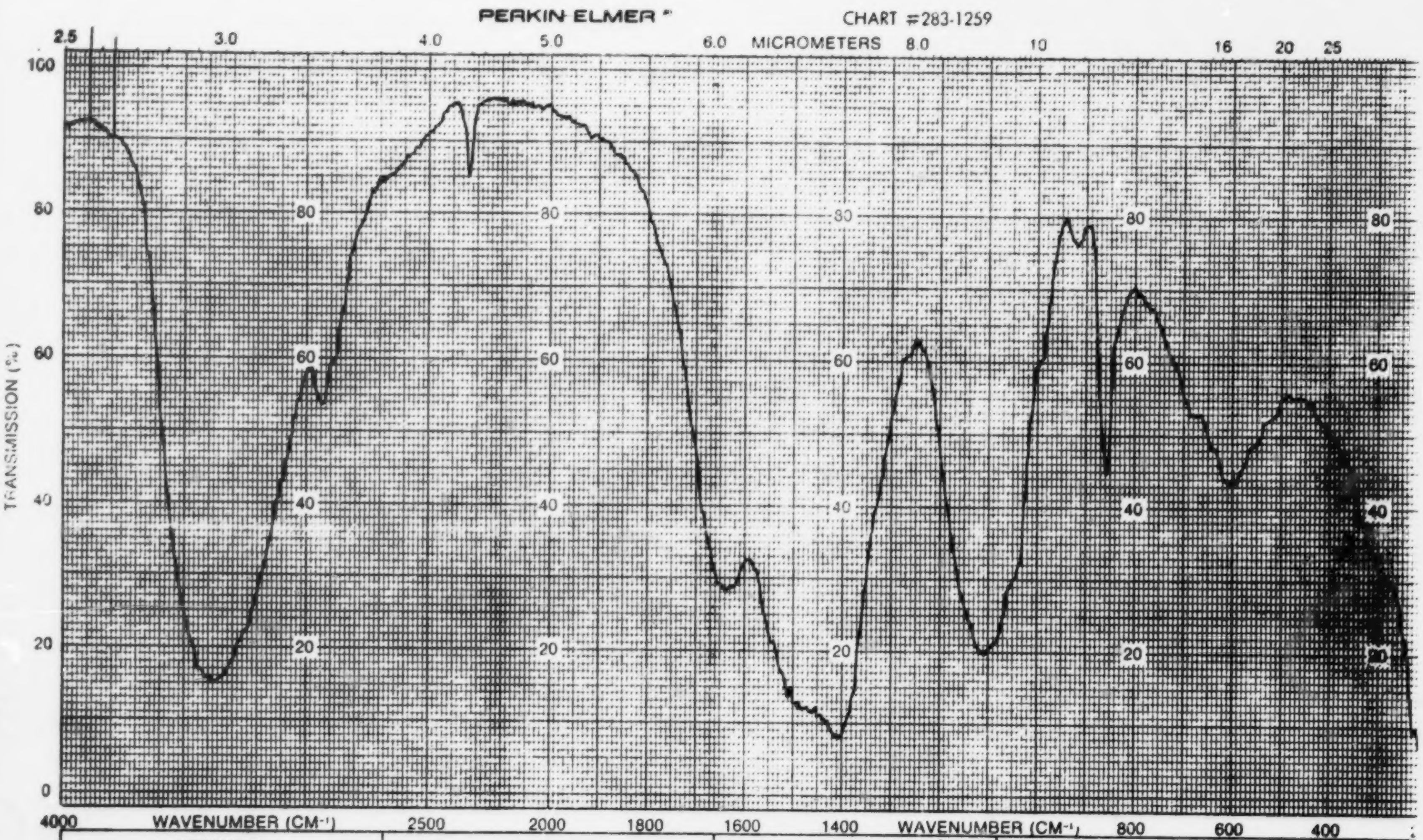


ABSCISSA
EXPANSION 1
SUPPRESSION
SAMPLE SH10
ORIGIN Salim Abbaud

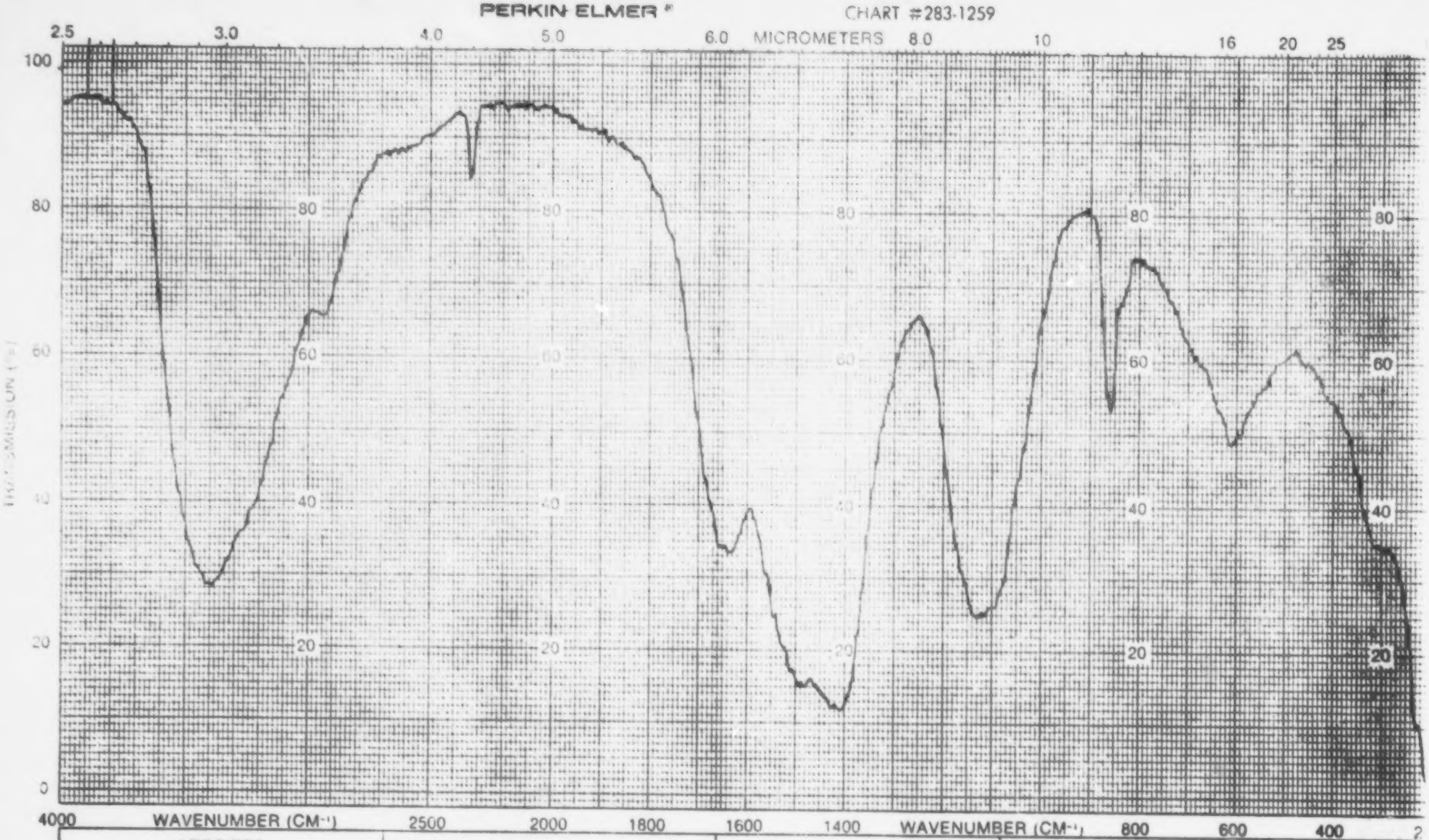
ORDINATE
EXPANSION 1
% T ✓ ABS
REMARKS 0.5% KBr

SCAN TIME 12 min
RESPONSE 1
SLIT PROGRAM N
SOLVENT
CONCENTRATION

REP. SCAN
TIME DRIVE
OPERATOR S.A.
SINGLE BEAM
PRE SAMPLE CHOP
DATE June 4
CELL PATH
REFERENCE 164-86 IR



ABSCISSA EXPANSION 1 SUPPRESSION	ORDINATE EXPANSION 1 % T ✓ ABS	SCAN TIME 12 min RESPONSE 1 SLIT PROGRAM N	REP. SCAN SINGLE BEAM TIME DRIVE PRE SAMPLE CHOP OPERATOR S.A. DATE June 4/
SAMPLE SH 30	REMARKS 0.5% KBr	SOLVENT	CELL PATH
ORIGIN Sami Abboud		CONCENTRATION	REFERENCE 165-861R



ABSCISSA
EXPANSION 1
SUPPRESSION

ORDINATE
EXPANSION '
% T ✓ ABS

SCAN TIME
RESPONSE
SLIT PROGRAM

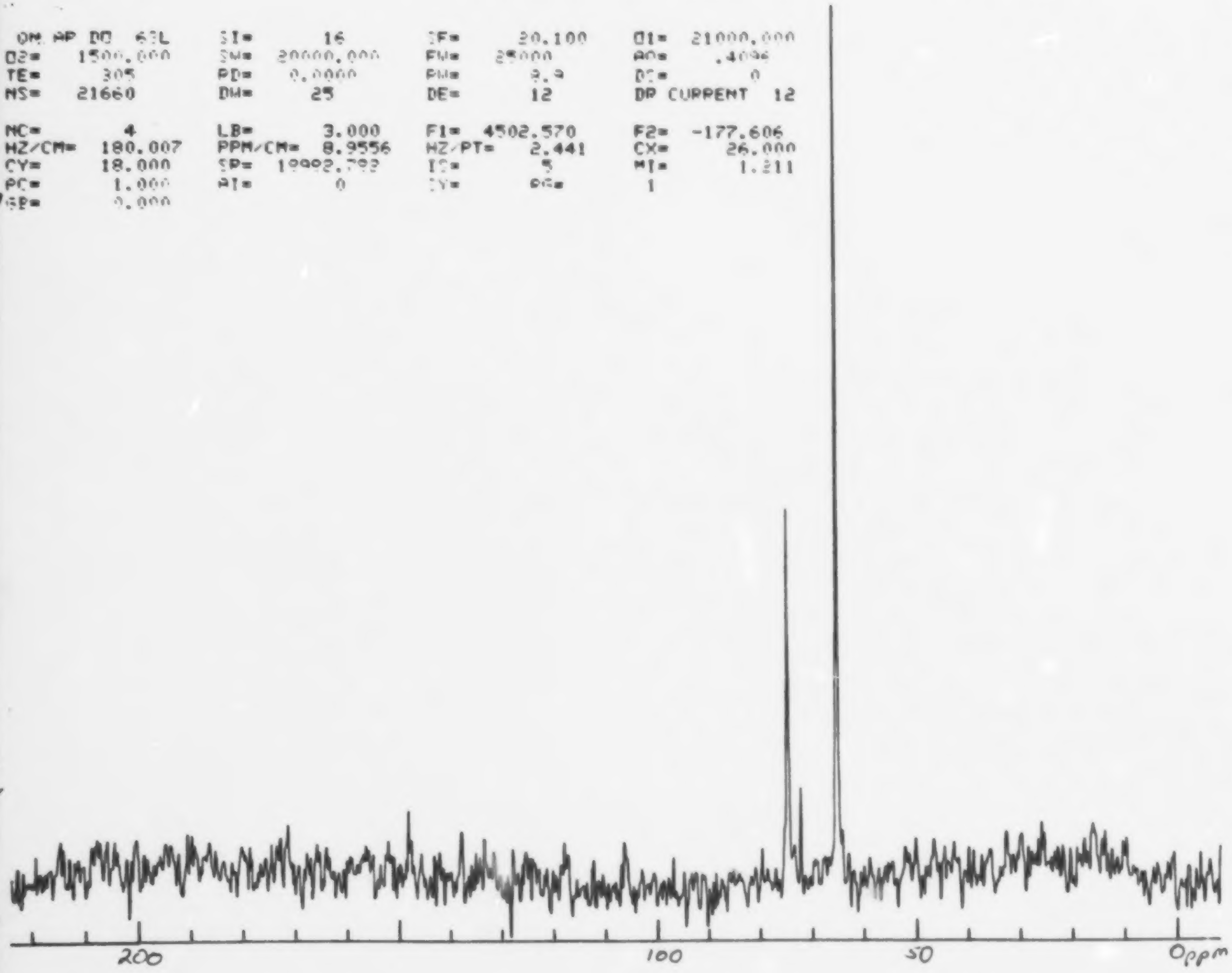
REP. SCAN
SINGLE BEAM
TIME DRIVE
PRE SAMPLE CHOP
OPERATOR S.A.
DATE June 4

SAMPLE SH100
ORIGIN Salim Abboud

REMARKS 0.5% KBr

SOLVENT
CONCENTRATION

CELL PATH
REFERENCE 166-86 IR



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NC=       4        LB=       3.000     F1=  4502.570      F2=  -177.606
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PC=      1.000     AI=       0         SY=      PG=        1
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UBK#17 No: 161:86

20.1 MHz ^{13}C

Sample

SH-1 (218 mg)

Solvent Aqm D_2O/2drops
10m NaOH

Tube size 10 mm

Temp. 306° K

Comments:
Ref SR=18982.782
Inverse gated
SW=20000
Delay = 2sec
Cr(AcAc)$_3$ added
LB = 3.0 Hz

NS = 21660 scans

Transform 16/8 K

Plot 180 Hz/cm

Date July 11, 1986
Operator W. Wade

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```
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O2=   1500.000   SW= 20000.000    RW=    25000        AQ=      .4096
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NS=    21660     DW=       25     DE=         12      DP CURRENT  12

NC=        2     LB=    50.000    F1=   4502.570      F2=   -177.606
HZ/CM= 180.007   PPM/CM= 8.9556   HZ/PT=   2.441      CX=     26.000
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GB=    0.000
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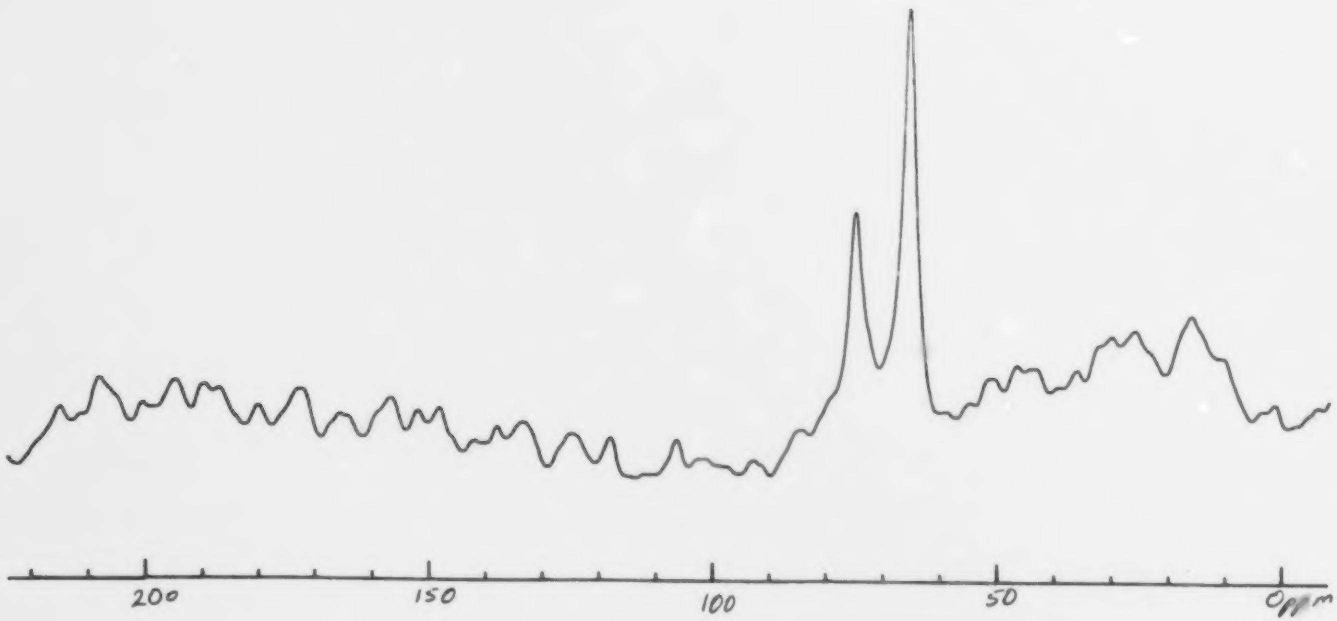

DISK#14

ABDOu086.161

No: 161-86.

20.1 MHz ¹³C

Sample

SH-1 (218 mg)

Solvent 2gm D₂O/2drops 10M NaOH

Tube size 10 mm

Temp. 30.6° K

Comments:

Ref SR = 18982.782

Inverse gated

SW = 20000

Delay =

Cr(AcAc) added

LB = 50.0 Hz

NS = 21660 scans

Transform 16/8 K

Plot 180 Hz / cm

Date July 11, 1986

Operator W. Wade

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NC=        4       LB=      3.000       F1=  4500.128        F2=   -177.606
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GF=       0.000
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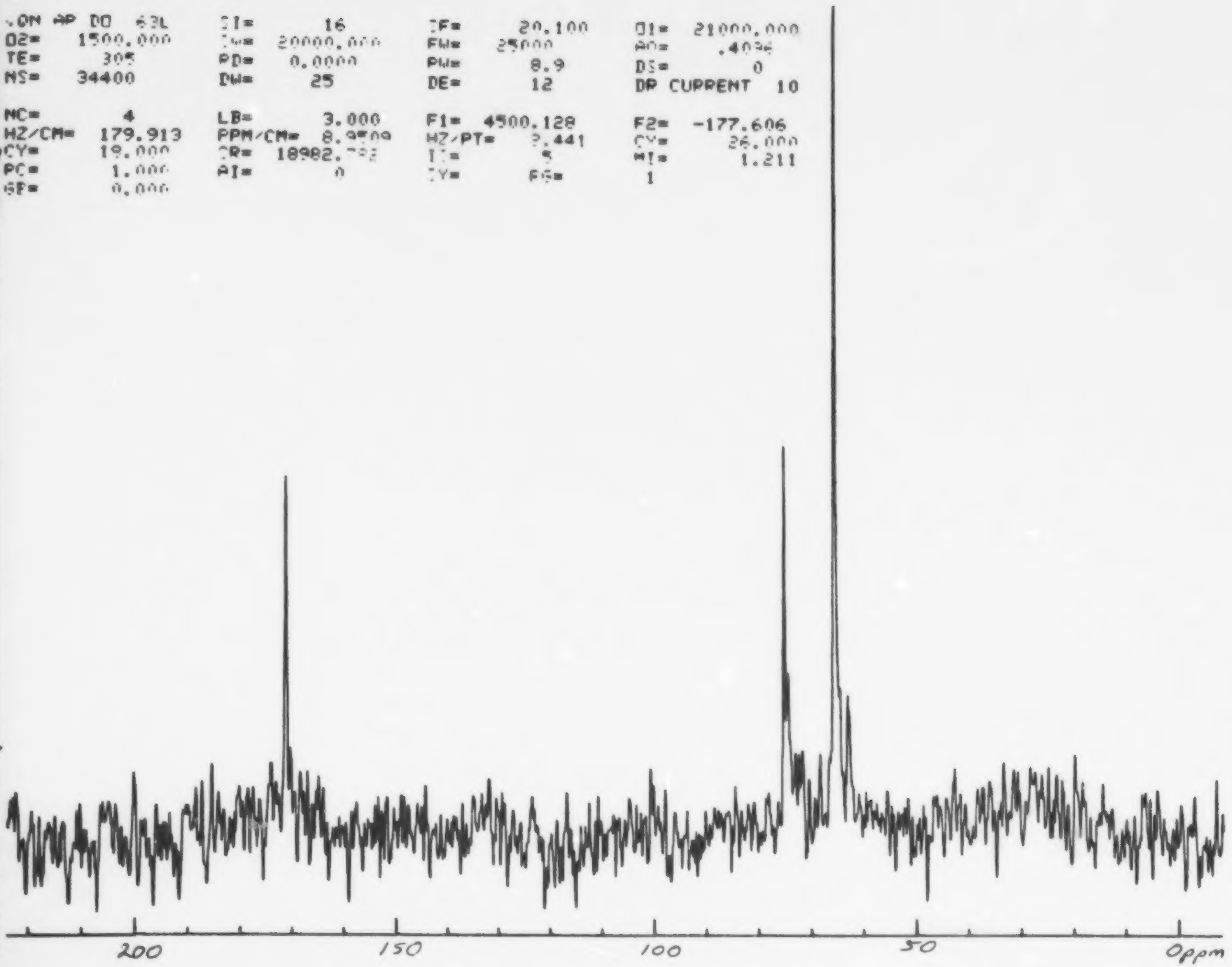

Disk #14. No: 160-86
ABOOW086.160

20.1 MHz 13C

Sample
SH-10 (65mg)

Solvent 1.4gm D2O/4 drops
10M NaOH

Tube size 10 mm

Temp. 306 K

Comments:
Ref SR = 18982.782
Inverse gated
SW = 20000
Delay = 2 sec
Cr(AcAc)3 added
LB = 3.0 Hz

NS = 34400 scans

Transform 16/8 K

Plot 180 Hz/cm

Date July 11, 1986

Operator W. Wade

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O2=   1500.000     SW=  20000.000       FW=   25000         AQ=      .4096
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NS=   34400        DW=      25          DE=       12        DP CURPENT  10

NC=        3       LB=      50.000      F1=  4500.128       F2=   -177.606
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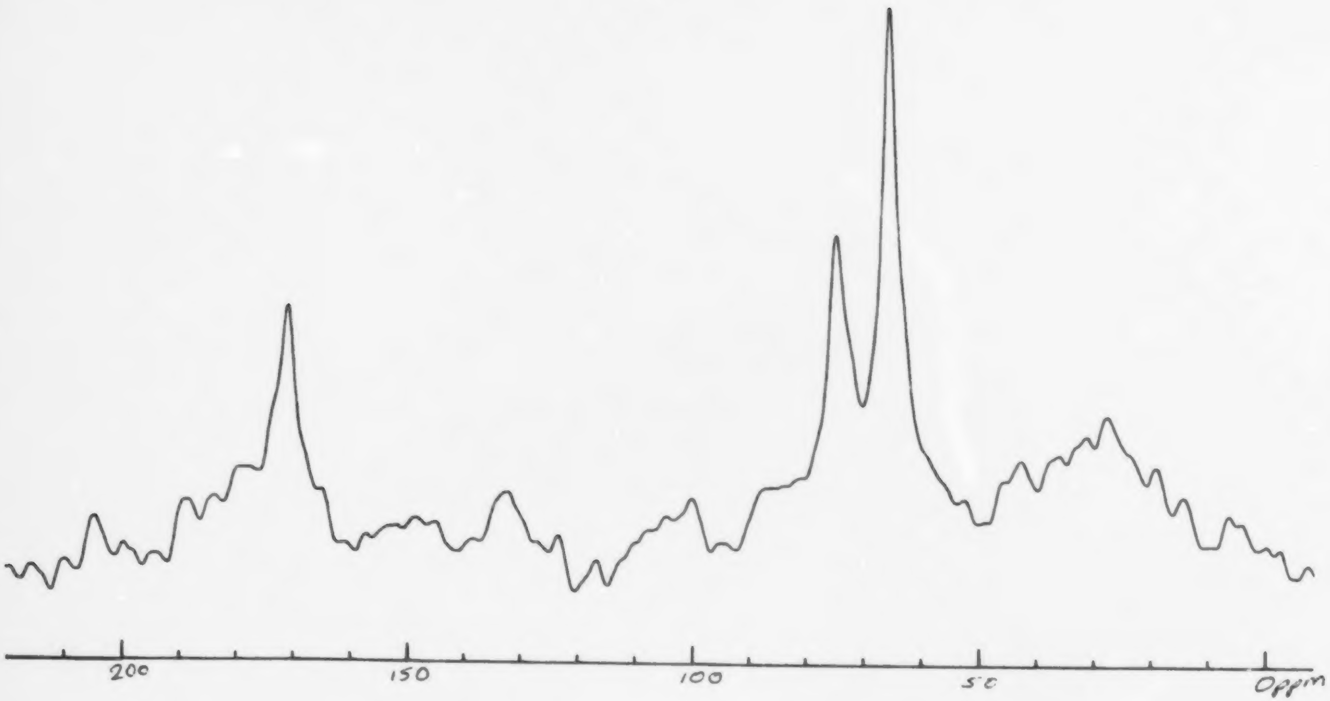

[illegible]

No: 16.Q-86
ABBOW086.160

20.1 MHz ^{13}C

Sample

SH-10 (65 mg).

Solvent 1.4mg D$_2$O/4 drops
10m NaOH

Tube size 10 mm

Temp. 306° K

Comments:
Ref SR= 18982.782
Inverse gated
SW= 20000
Delay = 2sec
Cr(AcAc)$_3$ added
LB= 50.0 Hz

NS = 34.400 scans

Transform 16/8 K

Plot 180 Hz/cm

Date July 11, 1986
Operator W. Wade

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NS=    20464        DW=       25         DE=       12          DP CURRENT  10

NC=        4        LB=         3.000    F1=  4500.128         F2=  -177.606
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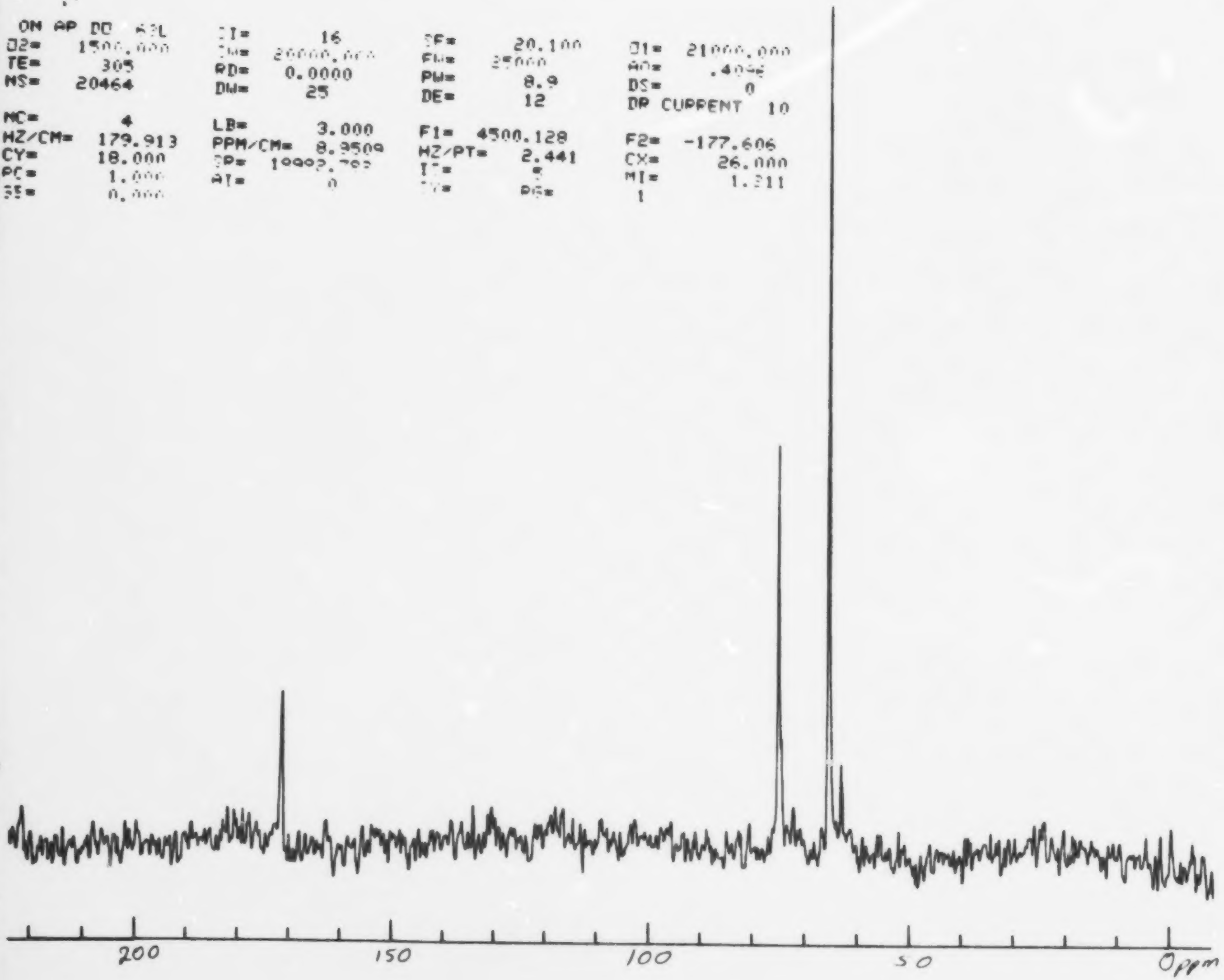

DISK#14 No:162-86

20.1 MHz ^{13}C

Sample
SH-30 (67mg).

Solvent 1gr. D₂O/2drops. 10M NaOH
Tube size 10 mm

Temp. 306° K

Comments:
Rf SR=18982.782
Inverse gated
SW=20000
Delay=2sec
Cr(AcAc)$_3$ added
LB=3Hz

NS = 20464 scans

Transform 16/8 K

Plot 180 Hz/cm

Date July 11, 1986
Operator W. Wade

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```
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O2=    1500.000     SW=  20000.000      FW=   25000         AO=     .4096
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NS=    20464        DW=       25        DE=       12        DR CURRENT  10

NC=        2        LB=      50.000     F1=  4500.128       F2=  -177.606
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GB=        0.000
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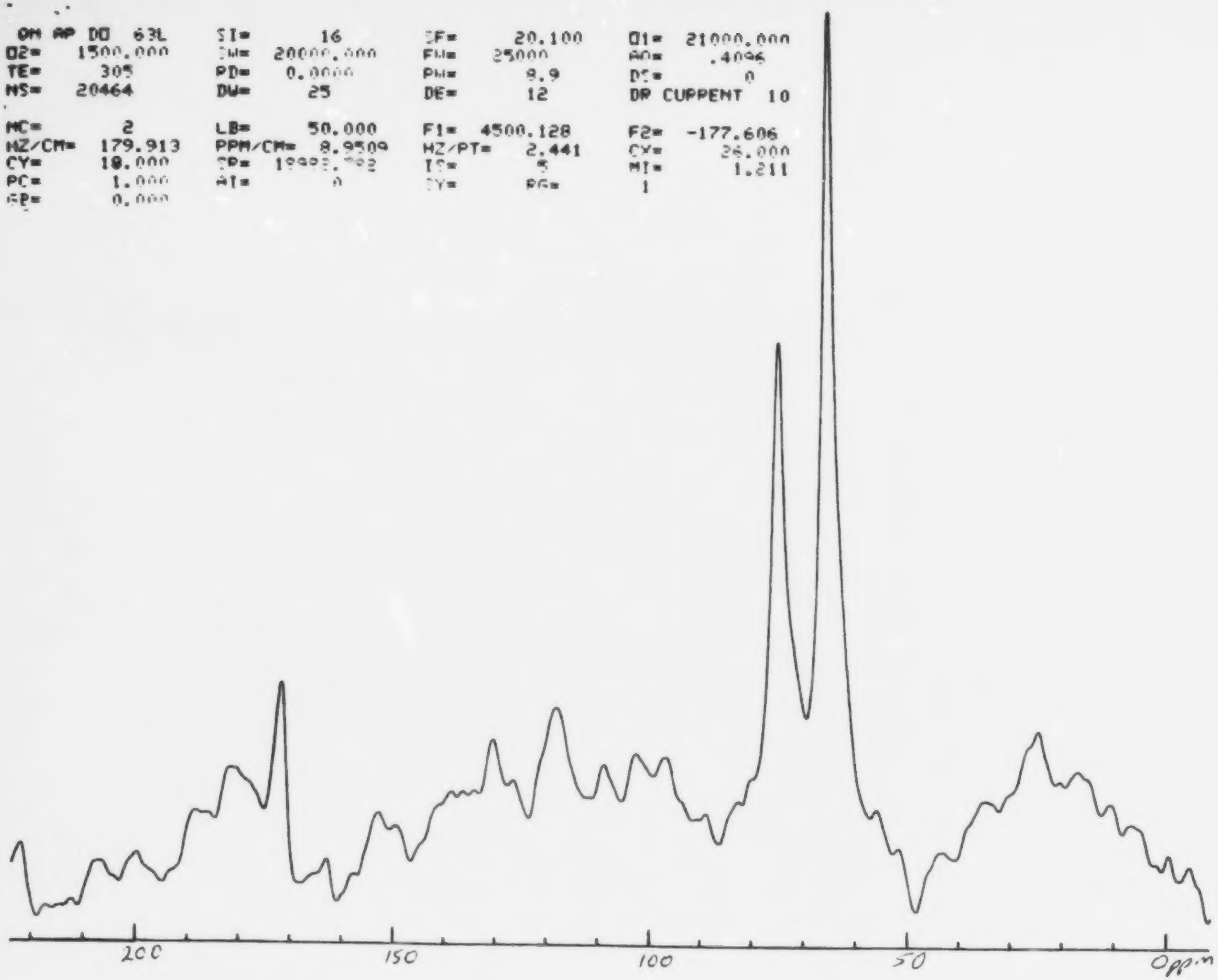

No: 142-86

20.1 MHz ^{13}C

Sample

SH-30 67mg

Solvent 1gm D_2O / 2drops 10M NaOH

Tube size 10 mm

Temp. 306° K

Comments:

Ref SR = 18982.782

Inverse gated

SW = 20000

Delay = 2sec

Cr(AcAc)$_3$ added

LB = 50 Hz

NS = 20464 scans

Transform 16/8 K

Plot 180 Hz / cm

Date July 11, 1986

Operator W. Wade

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NC=        1      LB=      3.000     F1=  4500.128      F2=  -177.606
HZ/CM=  179.913   PPM/CM=  8.9509    HZ/PT=   2.441     CY=     26.000
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PC=      1.000    AI=         0      CY=       RG=       1
GE=      0.000
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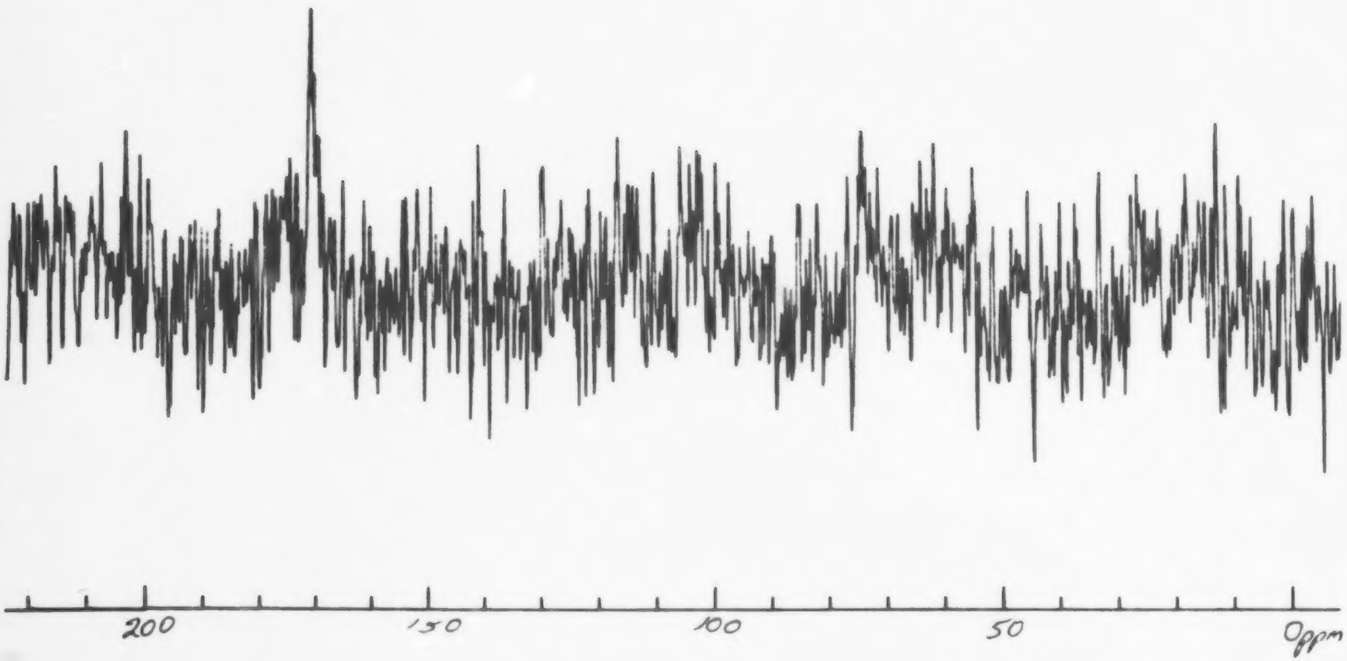


[illegible] No: 163-86

20.1 MHz ^{13}C

Sample
SH-100 (52 mg)

Solvent 1gm D_2O/2drops
10M NaOH
Tube size 10 mm

Temp. 305° K

Comments:
Rf SR=18982782
Inversegated
SW=20000
Delay = 2sec
$Cr(AcAc)_3$ added
LB = 3 Hz
NS = 26160 scans

Transform 16K K

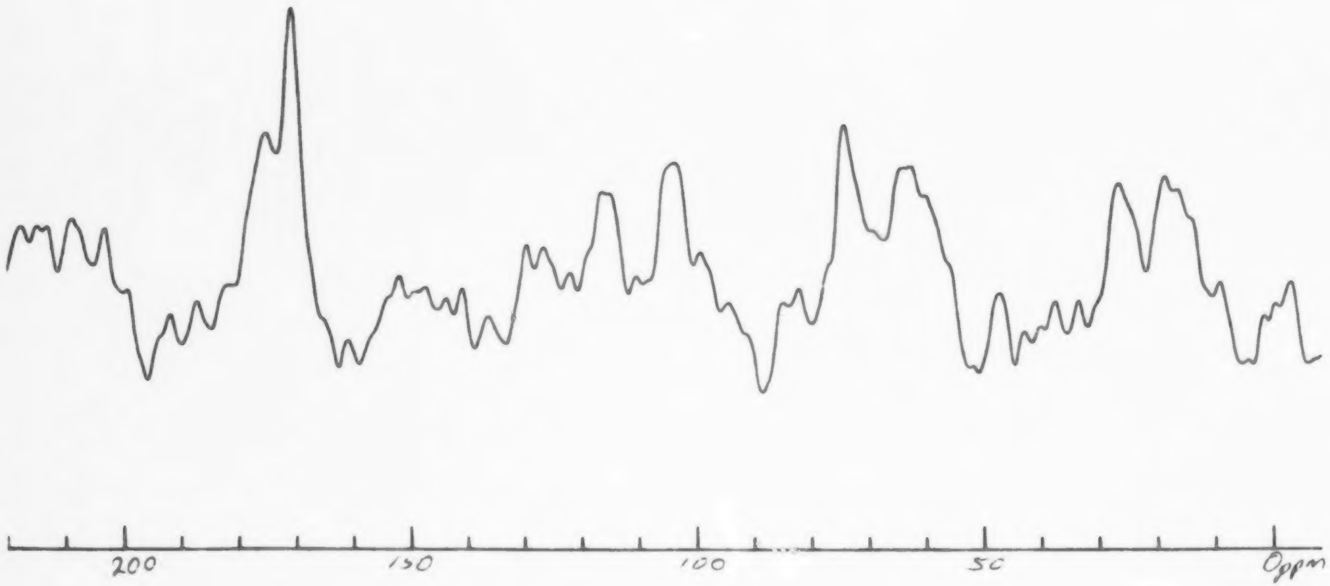
Plot 180 Hz/cm

Date July 11, 1986
Operator W. Wade

ALBERTA RESEARCH COUNCIL

```
 ON AP DO  62L      SI=        16        SF=      20.100     O1=  21000.000
O2=    1500.000     SW=  20000.000       FW=   25000          AQ=      .4096
TE=      305        PD=    0.0000        PW=        8.9      DS=          0
NS=   26160         DW=       25         DE=         12      DR CURRENT  10

NC=       1         LB=       50.000     F1=  4500.128       F2=  -177.606
HZ/CM=  179.913     PPM/CM=   8.9509     HZ/PT=     2.441    CX=     26.000
CY=      18.000     SR=  18982.782       IS=          5      MI=      1.211
FC=       1.000     AI=          0       DY=        FG=        1
GB=       0.000
```



DSK814

No: 163:86.

20.1 MHz ^{13}C

Sample
SH-100 (52 mg)

Solvent 1gm. 0.01. 2 drops
10M NaOH
Tube size 10 mm

Temp. 305° K

Comments:
Ref SR = 18982.782
Inverse gated
SW = 20000
Delay = 2 sec
Cr(A.Ac)$_3$
LB = 50 Hz

NS = 26160 scans

Transform 16/8 K

Plot 180 Hz/cm

Date July 11, 1986
Operator W. Wade

ALBERTA
RESEARCH
COUNCIL

Appendix D

Alberta Clean Water Act Licenses for Edmonton Water Treatment Plants

Alberta
ENVIRONMENT
Environmental Protection Services
Standards and Approvals Division
Municipal Engineering Branch

9820 106 Street Edmonton, Alberta Canada T5K 2J6 403 427 5877 Telex 037 2006 TWX 610-831 2636

March 26, 1985

Mayor and Council
City of Edmonton
City Hall
1 Sir Winston Churchill Square
EDMONTON, Alberta
T5J 2R7

Attention: C.J. McGonigle, City Clerk

Dear Sir:

RE: Facility: Rossdale Water Treatment Plant
Licence to Operate No: 78-ML-035 R2 (85)

We are enclosing the renewed Licence to Operate the above noted facility.

The purpose of this Licence is to clearly outline the operating and reporting requirements for your Rossdale Water Treatment Plant. We request that you review the attached terms and conditions with your operations staff, giving particular attention to these requirements. A copy of the Licence should be given to your operating staff for their continuing use.

If there are any questions about the terms and conditions of the Licence regarding the operation of the facility or the requirements for returns, please contact the Municipal Engineering Branch of the Pollution Control Division at 427-5837 in Edmonton. Representatives of the Pollution Control Division will be carrying out periodic inspections of the facility and will review your returns to ensure compliance with the operating requirements.

Please note that the Licence to Operate your Rossdale Water Treatment Plant expires on April 1, 1990. Application to renew the Licence should be made to the Standards and Approvals Division approximately three months prior to the expiry date.

Yours truly,

P. Kan

Prosper Kan, M.A.Sc., P. Eng.
Regional Engineer

c.c. L. Gyurek, Edmonton Water and Sanitation
Pollution Control Division

Alberta
ENVIRONMENT

LICENCE TO OPERATE OR USE

LICENSE NO. 78-ML-035 R2 (85)
PERMIT NO. 84-MP-153
FILE NO 85-MA-1001

TO Mayor

City of Edmonton

Pursuant to section 4 of the Clean Water Act, Revised Statutes of Alberta 1980
a licence to operate or use

Rossdale Water Treatment Plant

is hereby issued subject to the terms, conditions and requirements attached hereto

Edmonton March 26, 1985

S. L. Lupul
Acting Director of Standards and Approvals

LICENCE NO.
78-ML-035 R2 (85)

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

1. This Licence authorizes the operation of a water treatment plant located at 102 Street - 96 Avenue, known as the Rossdale Water Treatment Plant, which consists of addition of chlorine dioxide, lime softening, coagulation and flocculation, filtration, seasonal addition of activated carbon for taste and odour control, disinfection with combined chlorine, and fluoridation. Any major change to the process train or change in the method of disinfection at the plant, except changes associated with normal plant operation and maintenance or emergency situations, shall be approved in writing by the Director of Standards and Approvals.

2. This facility is classified as a WT IV in accordance with the Facility Classification Program as administered by the Director of Pollution Control.

4. The water treatment facility shall be maintained and operated to produce treated water which meets the *Guidelines for Canadian Drinking Water Quality, 1978* as amended by the Province of Alberta.

5. Disinfection of the water supply is required. The equipment and controls used for disinfection shall at all times be well maintained and properly operated. Spare parts needed to maintain equipment shall be readily accessible. A "combined chlorine" residual of at least 1.8 milligrams per litre shall be maintained in any treated water entering the distribution system after a contact time of at least 20 minutes, and a combined residual of at least 1.0 milligram per litre shall be maintained at all the reservoirs within the City limits. A combined chlorine residual of not less than 0.5 milligram per litre shall be maintained in all parts of the water distribution system within the City limits. The combined residual at the water treatment plant shall be tested and recorded at least once daily.

6. The Director of Pollution Control (c/o Regional Engineer, 5th Floor, Oxbridge Place, 9820 - 106 Street, Edmonton, Alberta) shall receive a copy of all analyses for bacteriological quality. The number of samples taken shall be a minimum of 146 per month in accordance with the latest edition of Guidelines for Canadian Drinking Water Quality or as specified by the Regional Engineer, Municipal Engineering Branch, Pollution Control Division. The samples shall be submitted so that they safely reach the Public Health Laboratory within 24 hours.

7. Water treatment plant control shall be maintained at all times to provide a fluoride ion concentration of 1.0 mg/L in the treated water. A monthly average variation from 1.0 mg/L of ± 0.1 mg/L and a daily variation of ± 0.2 mg/L is acceptable.

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

8. A monthly report outlining the following information shall be submitted to the Director of Pollution Control and to the Edmonton Local Board of Health by the fifteenth day of the month following the data month:

   a. The daily reading of the water meter which controls the fluoridation equipment or that which determines the amount of water to which the fluoride is added.
   b. The daily volume of water fluoridated.
   c. The daily weight of fluoride compound in the feeder.
   d. The daily weight of fluoride compound in stock.
   e. The daily weight of the fluoride compound fed to the water.
   f. The fluoride content of the raw and fluoridated water determined by laboratory analysis
      i) Treated water shall be analyzed daily.
      ii) Raw water shall be analyzed at least once a week.

9. A monthly report outlining the following information shall be submitted to the Director of Pollution Control by the fifteenth day of the month following the data month:

   a. Average daily flow of treated water produced at the plant.
   b. Peak daily flow of treated water produced at the plant.
   c. A copy of the chemical water analysis for at least one sample of raw and one sample of treated water each month.
   d. A summary of the volume of lime sludge produced at the Rossdale Water Treatment Plant, and the method and location of disposal of lime sludge wastes.
   e. A summary of the volumes of other waste streams returned to the North Saskatchewan River and their composition.
   f. A summary of the chemical additives and times utilized.
   g. A summary of any operational difficulties or repairs that were undertaken.

10. An annual report outlining the following information shall be submitted to the Director of Pollution Control before the 31st day of May of the year following the data year:

   a. A heavy metal analysis for at least one sample collected during the summer and one sample collected during the winter. Concentrations of Arsenic (As), Barium (Ba), Cadmium (Cd), Chromium (Cr^{+6}), Cyanide (CN), Lead (Pb), Mercury (Hg), Nickel (Ni), Selenium (Se), and Silver (Ag) shall constitute the heavy metal analysis.
   b. A summary of the average daily flow of treated water produced at the plant.

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

c. A summary of the peak daily flows of treated water produced at the plant.
d. A summary of the chemical water analysis.
e. A summary of the chemical additives and times utilized.
f. A summary of the volume of lime sludge disposed of and location and method of disposal.
g. A summary of any operational difficulties.
h. An overview of plant operation for the year including any expansion, major equipment replacements, or any process changes that were incorporated.

11. The Director of Pollution Control shall be notified when a change in the chief treatment plant operator occurs. The name, address, and qualifications of the new operator shall be submitted to the Director of Pollution Control.

12. All analytical procedures shall be in accordance with the latest edition of "Standard Methods for the Examination of Water and Waste Water", as published by the American Public Health Association, American Water Works Association, and the Water Pollution Control Federation, or by an alternative method approved by the Director of Standards and Approvals.

13. The issuance of this Licence does not restrict the authority of either the Director of Standards and Approvals or the Director of Pollution Control in carrying out his specific function in accordance with the Clean Water Act and the regulations issued thereunder.

14. This Licence is granted on the basis of current knowledge, technology and circumstances, and if deemed necessary by the Director of Standards and Approvals, the terms, conditions and requirements attached to this Licence may be revised or amended.

15. This Licence expires on April 1, 1990.

DATED March 26, 1985

S. L. Lupul
ACTING DIRECTOR

Alberta
ENVIRONMENT
Environmental Protection Services
Standards and Approvals Division
Municipal Engineering Branch

9820 - 106 Street, Edmonton, Alberta, Canada T5K 2J6 403/427-5877 Telex 037-2006 TWX 610-831-2636

March 26, 1985

Mayor and Council
City of Edmonton
City Hall
1 Sir Winston Churchill Square
EDMONTON, Alberta
T5J 2R7

Attention: C.J. McGonigle, City Clerk

Dear Sir:

RE: Facility: E.L. Smith Water Treatment Plant
Licence to Operate No: 78-ML-036 R2 (85)

We are enclosing the renewed Licence to Operate the above noted facility.

The purpose of this Licence is to clearly outline the operating and reporting requirements for your E.L. Smith Water Treatment Plant. We request that you review the attached terms and conditions with your operations staff, giving particular attention to these requirements. A copy of the Licence should be given to your operating staff for their continuing use.

If there are any questions about the terms and conditions of the Licence regarding the operation of the facility or the requirements for returns, please contact the Municipal Engineering Branch of the Pollution Control Division at 427-5837 in Edmonton. Representatives of the Pollution Control Division will be carrying out periodic inspections of the facility and will review your returns to ensure compliance with the operating requirements.

Please note that the Licence to Operate your E.L. Smith Water Treatment Plant expires on April 1, 1990. Application to renew the Licence should be made to the Standards and Approvals Division approximately three months prior to the expiry date.

Yours truly,

P. Kan

Prosper Kan, M.A.Sc., P. Eng.
Regional Engineer

c.c. L. Gyurek, Edmonton Water and Sanitation
Pollution Control Division

Alberta

ENVIRONMENT

LICENCE TO OPERATE OR USE

LICENSE NO. 78-ML-036 R2 (85)

PERMIT NO.

FILE NO. 85-MA-1002

TO Mayor

City of Edmonton

Pursuant to section 4 of the Clean Water Act, Revised Statutes of Alberta 1980
a licence to operate or use

E.L. Smith Water Treatment Plant

is hereby issued subject to the terms, conditions and requirements attached hereto.

Edmonton March 26, 1985

L. L. Lupul

Acting Director of Standards and Approvals

LICENCE NO.
78-ML-036 R2 (85)

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

1. This Licence authorizes the operation of a water treatment plant located at 170 Street - 34 Avenue, known as the E.L. Smith Water Treatment Plant, which consists of addition of chlorine dioxide, lime softening, coagulation and flocculation, filtration, seasonal addition of activated carbon for taste and odour control, disinfection with combined chlorine, and fluoridation. Any major change to the process train or change in the method of disinfection at the plant, except changes associated with normal plant operation and maintenance or emergency situations, shall be approved in writing by the Director of Standards and Approvals.

2. This facility is classified as a WT IV in accordance with the Facility Classification Program as administered by the Director of Pollution Control.

4. The water treatment facility shall be maintained and operated to produce treated water which meets the Guidelines for Canadian Drinking Water Quality, 1978 as amended by the Province of Alberta.

5. Disinfection of the water supply is required. The equipment and controls used for disinfection shall at all times be well maintained and properly operated. Spare parts needed to maintain equipment shall be readily accessible. A "combined chlorine" residual of at least 1.8 milligrams per litre shall be maintained in any treated water entering the distribution system after a contact time of at least 20 minutes, and a combined residual of at least 1.0 milligram per litre shall be maintained at all the reservoirs within the City limits. A combined chlorine residual of not less than 0.5 milligram per litre shall be maintained in all parts of the water distribution system within the City limits. The combined residual at the water treatment plant shall be tested and recorded at least once daily.

6. The Director of Pollution Control (c/o Regional Engineer, 5th Floor, Oxbridge Place, 9820 - 106 Street, Edmonton, Alberta) shall receive a copy of all analyses for bacteriological quality. The number of samples taken shall be a minimum of 146 per month in accordance with the latest edition of Guidelines for Canadian Drinking Water Quality or as specified by the Regional Engineer, Municipal Engineering Branch, Pollution Control Division. The samples shall be submitted so that they safely reach the Public Health Laboratory within 24 hours.

7. Water treatment plant control shall be maintained at all times to provide a fluoride ion concentration of 1.0 mg/L in the treated water. A monthly average variation from 1.0 mg/L of ± 0.1 mg/L and a daily variation of ± 0.2 mg/L is acceptable.

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

8. A monthly report outlining the following information shall be submitted to the Director of Pollution Control and to the Edmonton Local Board of Health by the fifteenth day of the month following the data month:

   a. The daily reading of the water meter which controls the fluoridation equipment or that which determines the amount of water to which the fluoride is added.
   b. The daily volume of water fluoridated.
   c. The daily weight of fluoride compound in the feeder.
   d. The daily weight of fluoride compound in stock.
   e. The daily weight of the fluoride compound fed to the water.
   f. The fluoride content of the raw and fluoridated water determined by laboratory analysis
      i) Treated water shall be analyzed daily.
      ii) Raw water shall be analyzed at least once a week.

9. A monthly report outlining the following information shall be submitted to the Director of Pollution Control by the fifteenth day of the month following the data month:

   a. Average daily flow of treated water produced at the plant.
   b. Peak daily flow of treated water produced at the plant.
   c. A copy of the chemical water analysis for at least one sample of raw and one sample of treated water each month.
   d. A summary of the volume of lime sludge produced at the E.L. Smith Water Treatment Plant, and the method and location of disposal of lime sludge wastes.
   e. A summary of the volumes of other waste streams returned to the North Saskatchewan River and their composition.
   f. A summary of the chemical additives and times utilized.
   g. A summary of any operational difficulties or repairs that were undertaken.

10. An annual report outlining the following information shall be submitted to the Director of Pollution Control before the 31st day of May of the year following the data year:

   a. A heavy metal analysis for at least one sample collected during the summer and one sample collected during the winter. Concentrations of Arsenic (As), Barium (Ba), Cadmium (Cd), Chromium (Cr^{+6}), Cyanide (CN), Lead (Pb), Mercury (Hg), Nickel (Ni), Selenium (Se), and Silver (Ag) shall constitute the heavy metal analysis.
   b. A summary of the average daily flow of treated water produced at the plant.

TERMS, CONDITIONS AND REQUIREMENTS ATTACHED TO LICENCE

c. A summary of the peak daily flows of treated water produced at the plant.
d. A summary of the chemical water analysis.
e. A summary of the chemical additives and times utilized.
f. A summary of the volume of lime sludge disposed of and location and method of disposal.
g. A summary of any operational difficulties.
h. An overview of plant operation for the year including any expansion, major equipment replacements, or any process changes that were incorporated.

11. The Director of Pollution Control shall be notified when a change in the chief treatment plant operator occurs. The name, address, and qualifications of the new operator shall be submitted to the Director of Pollution Control.

12. All analytical procedures shall be in accordance with the latest edition of "Standard Methods for the Examination of Water and Waste Water", as published by the American Public Health Association, American Water Works Association, and the Water Pollution Control Federation, or by an alternative method approved by the Director of Standards and Approvals.

13. The issuance of this Licence does not restrict the authority of either the Director of Standards and Approvals or the Director of Pollution Control in carrying out his specific function in accordance with the Clean Water Act and the regulations issued thereunder.

14. This Licence is granted on the basis of current knowledge, technology and circumstances, and if deemed necessary by the Director of Standards and Approvals, the terms, conditions and requirements attached to this Licence may be revised or amended.

15. This Licence expires on April 1, 1990.

DATED March 26, 1985

ACTING DIRECTOR

Appendix E

Alberta Treated Water Survey Results for Edmonton Plants - 1983 to 1986

Alberta Environment Treated Water Survey 1983												
LOCATION					1983	Edmonton						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
pH	7.9	7.7	7.9	8.2	8.2	7.9	7.8	8	8.3	8	8.4	8.4
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	0.08	0.04	0.1	0.13	0.12	<0.02	<0.02	<0.02	0.03	<0.02	<0.02
CALCIUM	42	48	36	24	28	26	23	32	26	32	27	27
HARDNESS T	154	169	135	93	111	110	90	125	106	113	117	117
POTASSIUM	0.9	0.7	1.1	1.4	0.9	0.7	1	0.8	0.7	0.6	0.7	0.7
NO2+NO3	0.1	0.1	0.1	<0.05	0.12	0.09	0.3	<0.05	<0.05	<0.05	<0.05	0.09
FLUORIDE	0.84	0.78	1.02	0.82	0.85	0.93	1.04	1	1.05	0.84	1.12	0.96
SULFATE	45	45	45	47	50	50	60	65	42	42	52	54
ALKALINITY	124	134	107	68	80	78	43	92	82	87	90	75
TC (PARTIC)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
TURBIDITY	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
CONDUCTIVITY	337	361	320	255	263	263	242	291	262	272	296	278
TDS	184	195	171	129	144	141	127	172	135	140	153	146
MAGNESIUM	12	12	11	8	10	11	8	11	10	8	12	12
SODIUM	5	5	7	6	5	5	6	5	4	3	5	5
SILICA	4.4	4.7	3.9	3.9	3.7	3.6	3.5	3.3	2.9	2.6	3.8	3.9
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	0.2	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	4	4	6	2	2	<1	2	3	3	2	2	2
BICARBONATE	151	163	130	83	97	95	53	112	99	106	105	88
DOC	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
DIC	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
MERCURY	N/A	N/A	N/A	<0.0001	N/A	N/A	N/A	N/A	<0.0001	N/A	<0.0001	<0.0001
SELENIUM	<0.0002	0.0004	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COPPER	0.225	0.077	0.412	0.086	0.057	0.039	0.159	0.049	0.037	0.515	0.012	0.006
CADMIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001	<0.001	<0.001	<0.001
ZINC	0.043	0.038	0.034	0.012	0.015	0.013	0.042	0.013	0.014	0.04	<0.001	0.005
CHROMIUM	0.006	0.006	<0.001	0.003	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.003
VANADIUM	0.003	0.003	0.003	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002
ALUMINUM	0.121	0.39	0.19	0.335	0.455	0.432	0.615	0.36	0.4	0.13	0.325	0.43
ARSENIC	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COBALT	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
NICKEL	0.005	0.007	0.006	<0.001	<0.001	0.005	<0.001	0.006	<0.001	0.003	0.006	<0.001
LEAD	0.004	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.004	0.03	0.005	0.014
MANGANESE	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
MOLYBDENUM	<0.001	<0.001	<0.001	<0.001	<0.001	0.008	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
TN (PARTIC)	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

LOCATION					1983	Edmonton						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.500	<0.500	<0.500	<0.500	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100
a-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Captan	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050
Dacthal	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
p,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800
Dieldrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Dursban	<0.040	<0.040	<0.040	<0.010	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Endrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Heptachlor	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Methoxychlor	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Mirex	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Perthane	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040

LOCATION					1983	Edmonton						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	4.3	<0.1	<0.1	<0.1
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromoform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	8	4	3	1	0.6	0.9	39	44	51	55	25	33
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	0.8	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	<10	<10	<10	2.4	<10	0.2	<10	24	<10	<10
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	0.2	<0.2	<0.2	0.2	0.1	0.3	<0.2	<0.2	<0.2
Toluene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.2	<0.1	<0.1	<0.1
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	0.7	<0.5	<0.5	<0.5	<0.5	<0.5
o-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.2	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1

LOCATION					1983	Edmonton						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS												
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP												
POLYNUCLEAR AROMATICS												
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

LOCATION					1983	Edmonton						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLOROORGANICS												
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS												
Benzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS												
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS												
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<1.0	<1.0	<1.0	<1.0	<1.0	2	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

Alberta Environment Treated Water Survey 1984												
LOCATION					1984	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
pH	8.3	8.2	8.2	8.3	8.7	7.9	8.1	8.1	8.2	8.3	8.4	8.3
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	0.03	<0.02	<0.02	0.05	<0.02	0.24	<0.02	<0.02	<0.02	<0.02	<0.02
CALCIUM	22	27	26	27	27	29	31	34	39	32	35	23
HARDNESS T	100	113	106	109	104	101	123	134	139	125	133	103
POTASSIUM	0.6	0.7	0.6	0.8	1.2	1.2	0.8	0.8	0.5	0.8	0.7	0.7
NO2+NO3	0.1	0.1	0.14	0.08	<0.05	0.16	<0.05	<0.05	<0.05	<0.05	<0.05	0.08
FLUORIDE	1.13	0.97	1.08	0.92	0.97	0.92	0.94	0.92	0.99	0.91	0.88	1.02
SULFATE	51	51	50	52	68	71	56	63	50	54	52	50
ALKALINITY	65	70	67	67	63	51	77	94	95	90	98	65
TC (PARTIC)	N/A	<0.01	N/A	0.23	0.36	0.49	0.36	0.22	0.95	0.77	0.21	0.66
TURBIDITY	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
CONDUCTIVITY	247	267	255	258	277	274	277	310	294	293	304	249
TDS	132	150	136	139	152	148	151	170	162	161	166	130
MAGNESIUM	11	11	10	10	9	7	11	12	10	11	11	11
SODIUM	5	7	4	4	6	5	2	<1.0	<1.0	6	5	4
SILICA	4.4	3.8	3.1	3.3	2.6	5.1	3.9	2	1.6	2.4	2.9	3
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	3	5	4	4	3	4	4	3	4	3	3	2
BICARBONATE	79	85	82	82	66	62	94	114	116	110	114	80
DOC	N/A	10	N/A	<0.4	2.1	4.1	1.5	0.5	0.9	2.1	0.7	0.5
DIC	N/A	19	N/A	14.5	12.4	13.4	17.5	22	22.1	20.6	24.1	13.9
MERCURY	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
SELENIUM	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	N/A	N/A	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COPPER	0.004	0.013	0.014	0.004	0.007	0.006	0.012	0.017	0.033	0.002	0.008	0.011
CADMIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	0.002	<0.001	<0.001	0.002	<0.001
ZINC	0.01	0.011	0.008	0.002	0.004	0.003	0.005	0.005	0.007	0.014	0.004	0.016
CHROMIUM	<0.001	0.005	0.003	<0.001	0.005	0.004	0.011	0.01	0.003	<0.001	0.005	0.005
VANADIUM	<0.002	<0.002	0.003	<0.002	<0.002	<0.002	0.007	0.009	<0.002	<0.002	0.006	<0.002
ALUMINUM	0.35	0.389	0.338	0.324	1.25	0.223	0.411	0.658	0.519	0.748	0.697	0.47
ARSENIC	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COBALT	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.006	0.006	<0.001	<0.001	0.004	<0.001
NICKEL	0.003	0.005	<0.001	0.006	0.005	0.006	0.007	0.008	<0.001	<0.001	0.006	0.006
LEAD	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
MANGANESE	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
MOLYBDENUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.008	0.006	<0.001	<0.001	0.004	0.004
TN (PARTIC)	N/A	0.06	N/A	<0.01	0.35	<0.01	0.03	0.02	<0.01	0.05	0.02	0.15

LOCATION					1984	Edmonton	Rossdale					
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CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100
a-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Captan	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050
Dacthal	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
p,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800
Dieldrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Dursban	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Endrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Heptachlor	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Methoxychlor	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Mirex	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Perthane	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1221	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Aroclor 1232	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1242	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1248	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1254	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1260	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1262	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1268	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05

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PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	<0.1	<0.1	5	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	3.6
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Bromoform	<5.0	<5.0	<5.0	<5.0	N/A	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	6.1	1.1	0.4	0.3	4	2.9	11	1.9	1.3	2.2	1.5	2.4
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	<10	<10	N/A	<10	<10	<10	<10	<10	<10	23
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	N/A	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	1
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	0.4	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	0.8	<0.5	<0.5	<0.5
o-Xylene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.4

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ORGANOPHOSPHORUS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aspon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Crufomate	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Dasanit	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00
DDVP	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
DEF	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Demeton	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00
Dimethoate	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Di-Syston	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00
Ethion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fenthion	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25
Fenitrothion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fonofos	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Guthian	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Malathion	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17
Methyl Parathion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Methyl Trithion	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25
Mevinphos	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Mocap	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Paraoxon	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Parathion	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Phorate	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Ronnel	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Tetrachlorvinphos	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
ORGANONITROGEN	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Atrazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Aminocarb	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00
Bromacil	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Cobex	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Cyanazine	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Diazinon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Dinoseb	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Metribuzin	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Prometryne	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50
Propanil	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70
Propazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Simazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Terbacil	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Trifluralin (Treflan)	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03

LOCATION					1984	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS												
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP												
POLYNUCLEAR AROMATICS												
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

LOCATION					1984	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLOROORGANICS												
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS												
Benzidine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS												
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS												
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<1.0	2	1.5	1.2	N/A	<1.0	<1.0	<1.0	1	2.8	<1.0	<1.0

Alberta Environment
Treated Water Survey 1984

LOCATION					1984	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
pH	8.3	8.4	8.7	8.3	8.2	7.9	8.5	8.5	8.3	8.5	8.2	8.4
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	0.04	0.02	0.03	0.03	<0.02	<0.02	0.03	<0.02	<0.02	<0.02	<0.02	<0.02
CALCIUM	21	25	26	28	33	33	27	28	29	30	25	27
HARDNESS T	98	104	106	107	119	124	113	111	109	116	108	113
POTASSIUM	0.6	0.5	0.6	0.8	1.1	1.2	0.8	0.7	0.5	0.7	0.7	0.6
NO2+NO3	0.08	0.12	0.12	0.06	<0.05	0.07	<0.05	<0.05	<0.05	<0.05	<0.05	0.08
FLUORIDE	0.27	1	1.12	0.93	0.94	0.97	1	0.92	0.96	0.94	0.94	0.96
SULFATE	57	50	47	58	68	86	69	71	59	62	52	48
ALKALINITY	67	76	72	62	70	48	49	55	58	62	70	75
TC (PARTIC)	N/A	0.11	N/A	0.27	0.29	N/A	0.7	0.18	0.84	0.06	0.34	0.54
TURBIDITY	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
CONDUCTIVITY	252	254	244	262	291	303	259	267	249	267	245	253
TDS	136	138	133	140	162	169	144	147	136	148	137	139
MAGNESIUM	11	10	10	9	9	10	11	10	9	10	11	11
SODIUM	4	4	3	4	6	6	3	<1.0	<1.0	5	4	4
SILICA	3.4	3.3	3	3.2	2.9	5.6	4	2.8	2.2	3.1	2.6	2.6
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	2	3	3	3	3	4	3	3	3	3	2	3
BICARBONATE	81	89	82	75	86	58	55	61	71	71	85	91
DOC	N/A	0.5	N/A	<0.4	1.9	N/A	1.3	<0.4	0.8	1	0.7	1
DIC	N/A	19	N/A	12.8	15.9	N/A	10.8	12.7	12.6	13.3	16.9	16.3
MERCURY	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.0002	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
SELENIUM	0.0003	<0.0002	<0.0002	<0.0002	<0.0002	N/A	N/A	<0.0002	<0.0002	<0.0002	0.0003	<0.0002
COPPER	0.002	0.004	0.002	0.003	0.002	0.107	0.037	0.008	0.003	0.005	0.004	0.006
CADMIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.003	0.002	<0.001	0.002	<0.001	<0.001
ZINC	0.002	<0.001	0.003	<0.001	0.002	0.002	0.004	<0.001	<0.001	0.011	0.002	0.011
CHROMIUM	<0.001	<0.001	0.005	<0.001	<0.001	0.004	0.01	0.011	0.005	0.004	<0.001	0.005
VANADIUM	<0.002	<0.002	0.003	<0.002	<0.002	0.003	0.01	0.009	<0.002	0.003	0.003	<0.002
ALUMINUM	0.62	0.514	0.722	0.324	0.361	0.165	0.278	0.38	0.519	0.317	0.551	0.68
ARSENIC	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	0.0003	<0.0002
COBALT	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.007	0.006	<0.001	<0.001	<0.001	0.003
NICKEL	0.004	0.004	<0.001	0.008	<0.001	<0.001	0.009	0.009	0.006	<0.001	0.003	0.005
LEAD	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
MANGANESE	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
MOLYBDENUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.007	<0.001	<0.001	<0.001	0.004
TN (PARTIC)	N/A	<0.01	N/A	<0.01	0.39	N/A	0.27	0.05	0.17	<0.01	0.06	0.03

LOCATION					1984	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100
a-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Captan	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050
Dacthal	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
p,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800
Dieldrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Dursban	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Endrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Heptachlor	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Methoxychlor	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Mirex	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Perthane	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1221	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Aroclor 1232	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1242	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1248	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1254	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1260	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1262	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1268	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05

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DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
ORGANOPHOSPHORUS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aspon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Crufomate	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Dasanit	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00
DDVP	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
DEF	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Demeton	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00
Dimethoate	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Di-Syston	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00
Ethion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fenthion	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25
Fenitrothion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fonofos	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Guthian	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Malathion	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17
Methyl Parathion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Methyl Trithion	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25
Mevinphos	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Mocap	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Paraoxon	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Parathion	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Phorate	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Ronnel	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Tetrachlorvinphos	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
ORGANONITROGEN	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Atrazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Aminocarb	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00
Bromacil	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Cobex	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Cyanazine	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Diazinon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Dinoseb	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Metribuzin	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Prometryne	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50
Propanil	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70
Propazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Simazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Terbacil	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Trifluralin (Treflan)	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03

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DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	<0.1	<0.1	<0.1	0.3	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	N/A
Bromoform	<5.0	<5.0	<5.0	<5.0	N/A	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	N/A
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	0.8	N.D.	N.D.	0.5	N/A	6	1.2	1.4	0.7	0.9	1.5	N/A
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	0.3	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	2	<10	N/A	<10	<10	<10	<10	<10	<10	<10
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	N/A	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	N/A	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	0.6	<0.5	<0.5	<0.5	N/A	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
o-Xylene	<0.1	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	0.5	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	0.5	<0.1	<0.1	<0.1	N/A	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1

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DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS												
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentachlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP												
POLYNUCLEAR AROMATICS												
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

					1984	E.L. Smith						
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLOROORGANICS												
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS												
Benzidine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS												
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS												
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	N/A	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<1.0	4.5	3.5	<1.0	N/A	1	<1.0	<1.0	1.5	1	<1.0	<1.0

Alberta Environment												
Treated Water Survey 1985												
LOCATION					**1985**	**Edmonton**	**E.L. Smith**					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
pH	8.4	8.2	8.3	8.2	8.3	8.1	8.5	9.2	8.2	8.4	8	8.2
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	<0.02	<0.05	<0.02	<0.06	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
CALCIUM	24	24	23	34	31	29	28	28	73	25	24	25
HARDNESS T	109	105	106	118	110	118	111	115	228	108	109	112
POTASSIUM	0.7	0.6	0.7	2.1	1.1	0.7	0.7	0.8	1.1	0.9	0.8	0.7
NO2+NO3	0.1	0.1	0.1	0.1	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	0.1
FLUORIDE	1.07	1.05	1.02	0.87	1	1	1	1.04	0.96	1.02	1.03	0.97
SULFATE	56	54	50	86	60	56	52	54	102	42	46	42
ALKALINITY	72	64	68	47	60	59	62	68	120	74	72	67
TC (PARTIC)	0.42	0.05	0.45	0.37	0.17	0.64	0.17	0.19	0.12	0.07	0.37	0.04
TURBIDITY	<0.1	<0.1	<0.1	<0.1	0.5	0.5	0.5	0.5	<0.1	<0.1	0.2	0.1
CONDUCTIVITY	261	242	246	301	279	254	252	263	453	248	254	245
TDS	142	134	133	172	149	140	136	143	272	135	135	128
MAGNESIUM	12	11	11	8	8	11	10	11	11	11	12	12
SODIUM	4	3	4	6	8	4	5	5	6	6	5	4
SILICA	3.4	2.8	2.9	3.1	2.4	2.3	2.7	3	4.8	3.6	3.4	3.1
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	2	3	3	7	5	4	3	3	6	5	4	3
BICARBONATE	85	78	82	57	73	72	72	82	147	86	88	82
DOC	<0.4	0.6	0.6	1.8	1.4	2	2.7	0.8	3.2	2.6	2.2	0.8
DIC	15.8	14.3	12.7	12.1	14.1	17.1	15.7	19.1	28.5	16.2	15.5	14.6
MERCURY	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
SELENIUM	0.0003	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COPPER	<0.001	0.007	0.006	0.004	0.006	0.006	0.01	0.006	<0.001	0.003	0.005	0.005
CADMIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
ZINC	0.007	<0.001	0.006	<0.001	0.015	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001
CHROMIUM	<0.001	<0.001	0.003	0.003	0.005	0.003	0.002	0.005	<0.001	<0.001	0.005	0.004
VANADIUM	<0.002	<0.002	0.003	<0.002	0.004	0.003	<0.002	0.005	<0.002	<0.002	<0.002	0.003
ALUMINUM	0.68	0.448	0.435	0.349	1.08	0.447	0.491	0.505	0.404	0.741	0.369	0.454
ARSENIC	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	0.0005	0.0005	0.0005
COBALT	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
NICKEL	<0.001	<0.001	0.003	0.004	0.004	0.003	0.003	0.006	<0.001	<0.001	0.006	0.005
LEAD	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.004	<0.003	<0.003	<0.003
MANGANESE	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
MOLYBDENUM	<0.001	<0.001	<0.001	<0.001	<0.001	0.008	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
TN (PARTIC)	0.11	N/A	0.04	<0.01	0.15	0.08	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01

LOCATION					1985	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.500	<0.500	<0.500	<0.500	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100
a-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Captan	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050
Dacthal	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
p,p'-DDT	<0.020	<0.020	<0.020	<0.800	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.010	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800
Dieldrin	<0.010	<0.010	<0.010	<0.040	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Dursban	<0.040	<0.040	<0.040	<0.010	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Endrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Heptachlor	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	0.41	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Methoxychlor	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Mirex	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Perthane	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1221	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Aroclor 1232	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1242	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1248	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1254	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1260	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1262	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1268	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05

LOCATION					1985	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	Trace	<0.1	<0.1	<0.1	<0.1
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	Trace	<0.5	Trace	Trace	Trace
Bromoform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	1.9	2.5	2.5	2.1	2	4.3	2.3	3.1	2.6	2	1.9	1.9
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	<10	<10	<10	<10	<10	<10	<10	Trace	<10	<10
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.2	Trace	<0.1	<0.1	<0.1	<0.1
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	Trace	<0.5	<0.5
o-Xylene	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.9	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	Trace	<0.1	Trace	<0.1	<0.1

LOCATION					1985	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
ORGANOPHOSPHORUS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aspon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Crufomate	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Dasanit	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00
DDVP	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
DEF	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Demeton	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00
Dimethoate	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Di-Syston	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00
Ethion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fenthion	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25
Fenitrothion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fonofos	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Guthian	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Malathion	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17
Methyl Parathion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Methyl Trithion	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25
Mevinphos	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Mocap	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Paraoxon	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Parathion	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Phorate	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Ronnel	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Tetrachlorvinphos	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
ORGANONITROGEN	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Atrazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Aminocarb	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00
Bromacil	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Cobex	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Cyanazine	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Diazinon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Dinoseb	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Metribuzin	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Prometryne	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50
Propanil	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70
Propazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Simazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Terbacil	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Trifluralin (Treflan)	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03

LOCATION					1985	Edmonton	E.L. Smith					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS												
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP												
POLYNUCLEAR AROMATICS												
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

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DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLOROORGANICS												
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	Trace	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS												
Benzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS												
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS												
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0*	<1.0*	<1.0	<1.0*	<1.0	Trace	Trace	<1.0	Trace	Trace	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0*	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<1.0*	<1.0*	<1.0*	<1.0	<1.0*	<1.0*	2.5	1.8	6	2.9	2	3.9
CHLORINATED HERBICIDE ACIDS												
Two,4-D	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Dicamba	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Dichlofop-methyl (Hoe Grass)	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
MCPA	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Picloram	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30
Two,4,5-T	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20

Alberta Environment												
Treated Water Survey 1985												
LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
pH	8.1	8.3	8.2	8	8.4	8.3	8.1	8.2	8.3	8.3	8	7.7
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	<0.02	<0.02	0.05	0.04	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02
CALCIUM	26	23	25	26	27	32	23	25	40	33	24	24
HARDNESS T	114	107	104	102	109	121	94	108	141	132	109	109
POTASSIUM	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8	0.8
NO2+NO3	0.1	<0.05	0.1	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	0.1
FLUORIDE	0.93	0.98	1.02	0.86	1	1.03	0.99	1.02	0.9	0.97	0.94	0.94
SULFATE	60	56	60	82	60	62	66	56	84	42	56	46
ALKALINITY	71	59	57	34	63	61	44	59	60	95	71	63
TC (PARTIC)	0.7	0.44	0.61	0.6	0.21	0.39	0.22	0.8	0.07	0.08	0.34	0.2
TURBIDITY	<0.1	<0.1	1.5	<0.1	0.5	0.5	0.5	0.5	<0.01	<0.1	0.2	0.1
CONDUCTIVITY	288	248	300	267	263	273	246	256	312	293	274	251
TDS	157	135	159	150	145	151	134	137	181	155	144	130
MAGNESIUM	12	12	12	12	12	12	12	12	12	12	12	12
SODIUM	5	5	5	5	5	5	5	5	5	5	5	5
SILICA	4.1	3.6	3.3	4.4	2.8	2.1	2	2.5	5.3	3.9	3.8	4.2
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	7	4	16	5	5	4	4	5	5	5	4	4
BICARBONATE	87	72	69	41	76	74	54	71	73	116	86	77
DOC	<0.04	<0.4	2.2	2.3	1	2.9	0.8	0.8	3.5	3.1	1.5	1.3
DIC	15.8	13.9	10.4	9.1	16.5	16.9	10.6	30.6	13.2	22.7	15.2	14.3
MERCURY	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
SELENIUM	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COPPER	0.006	0.011	0.011	0.1	0.013	0.009	0.007	0.112	0.003	0.012	0.007	0.006
CADMIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.002	<0.001	<0.001	<0.001	0.002
ZINC	0.012	<0.001	0.017	0.009	0.01	<0.001	<0.001	0.004	0.002	0.005	0.004	0.005
CHROMIUM	<0.001	<0.001	0.003	<0.001	<0.001	0.003	0.002	0.005	<0.001	<0.001	0.003	0.005
VANADIUM	<0.002	<0.002	<0.002	<0.002	<0.002	0.003	<0.002	0.004	<0.002	<0.002	<0.002	0.004
ALUMINUM	0.34	0.369	0.4	0.248	0.447	0.524	0.834	0.493	0.238	0.492	0.033	0.336
ARSENIC	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
COBALT	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	0.003	<0.001	<0.001	<0.001	0.003
NICKEL	<0.001	0.003	0.005	0.003	0.004	0.003	0.004	0.004	<0.001	<0.001	<0.001	0.006
LEAD	<0.003	<0.003	<0.003	0.005	<0.003	<0.003	<0.003	<0.003	0.004	<0.003	<0.003	<0.003
MANGANESE	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001
MOLYBDENUM	<0.001	<0.001	<0.001	<0.001	<0.001	0.008	<0.001	<0.001	<0.001	<0.001	<0.001	0.006
TN (PARTIC)	0.04	0.16	0.25	0.05	<0.01	0.06	<0.01	<0.01	<0.01	0.03	<0.01	<0.01

LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.500	<0.500	<0.500	<0.500	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100	<0.100
a-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Captan	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050	<0.050
Dacthal	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
p,p'-DDT	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800	<0.800
Dieldrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Dursban	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Endrin	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Heptachlor	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020	<0.020
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Methoxychlor	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030	<0.030
Mirex	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010	<0.010
Perthane	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300	<0.300
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200	<0.200
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040	<0.040
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1221	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Aroclor 1232	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1242	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1248	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1254	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Aroclor 1260	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1262	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Aroclor 1268	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05

LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	Trace	<0.1	<0.1	<0.1	<0.1
Bromodichloromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	Trace	<0.5	Trace	0.2	Trace
Bromoform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	3.5	<0.1	3.3	2.8	2.8	2.6	3.2	1	2.5	3.3	1.6	2.1
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	1.5	<0.1	0.8	1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	<10	Trace
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	Trace	Trace	<0.5	<0.5
o-Xylene	<0.1	<0.1	3.1	12	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	Trace
m-Xylene	0.4	0.4	0.1	12	0.4	<0.1	<0.1	0.4	<0.1	<0.1	<0.1	0.3
p-Xylene	<0.1	<0.1	1.5	12	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1

LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
ORGANOPHOSPHORUS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aspon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Crufomate	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Dasanit	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00	<4.00
DDVP	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
DEF	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Demeton	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00	<7.00
Dimethoate	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Di-Syston	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00	<25.00
Ethion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fenthion	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25	<1.25
Fenitrothion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Fonofos	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Guthian	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00	<2.00
Malathion	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17	<0.17
Methyl Parathion	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Methyl Trithion	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25	<0.25
Mevinphos	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Mocap	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Paraoxon	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Parathion	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06	<0.06
Phorate	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Ronnel	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Tetrachlorvinphos	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
ORGANONITROGEN	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Atrazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Aminocarb	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00	<100.00
Bromacil	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
Cobex	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Cyanazine	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Diazinon	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50	<0.50
Dinoseb	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15	<0.15
Metribuzin	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03
Prometryne	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50	<1.50
Propanil	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70	<0.70
Propazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Simazine	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00	<1.00
Terbacil	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10	<0.10
Trifluralin (Treflan)	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03	<0.03

LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS												
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP												
POLYNUCLEAR AROMATICS												
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0*	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

LOCATION					1985	Edmonton	Rossdale					
DATE	JANUARY	FEBRUARY	MARCH	APRIL	MAY	JUNE	JULY	AUGUST	SEPTEMBER	OCTOBER	NOVEMBER	DECEMBER
CHLOROORGANICS												
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS												
Benzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS												
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS												
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0*	<1.0*	<1.0*	<1.0	<1.0	<1.0*	<1.0	Trace	<1.0	Trace	Trace	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0*	<1.0	<1.0	<1.0	4	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	3	<1.0*	<1.0*	<1.0*	<1.0*	7.8	2.1	5	4.3	<1.0	2	1.3
CHLORINATED HERBICIDE ACIDS												
Two,4-D	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Dicamba	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Dichlofop-methyl (Hoe Grass)	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
MCPA	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20
Picloram	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30	<0.30
Two,4,5-T	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20	<0.20

Alberta Environment						
Treated Water Survey 1986						
LOCATION	**< 1986**	**Edmonton**	**E.L.Smith**	**W.T.P. >**	**E.L. Smith W.T.P.**	**18110 - 98 Ave., Edm.**
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	3-Mar-86
pH	8	7.9	8.1	N/A	8.1	N/A
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	0.36	<0.02	N/A	<0.02	N/A
CALCIUM	26	25	28	N/A	40	N/A
HARDNESS T	122	116	115	N/A	141	N/A
POTASSIUM	0.8	0.7	2.2	N/A	4.8	N/A
NO2+NO3	0.1	0.1	0.11	N/A	0.2	N/A
FLUORIDE	0.96	0.99	0.95	N/A	0.95	N/A
SULFATE	52	54	74	N/A	95	N/A
ALKALINITY	72	71	62	N/A	55	N/A
TC (PARTIC)	N/A	0.36	0.17	N/A	0.1	0.07
TURBIDITY	<0.1	0.1	0.1	N/A	<0.1	N/A
CONDUCTIVITY	259	261	286	N/A	353	N/A
TDS	143	143	166	N/A	200	N/A
MAGNESIUM	14	13	11	N/A	10	N/A
SODIUM	5	5	9	N/A	8	N/A
SILICA	3.9	3.6	4	N/A	2.9	N/A
NITRITE	<0.05	<0.05	<0.05	N/A	<0.05	N/A
CHLORIDE	2	2	4	N/A	8	N/A
BICARBONATE	88	87	76	N/A	67	N/A
DOC	0.8	1.8	1.4	N/A	6.6	7.3
DIC	15.6	17.1	14.1	N/A	11.9	13.5
MERCURY	<0.0001	<0.0001	<0.0001	N/A	N/A	N/A
SELENIUM	<0.0002	<0.0002	<0.0002	N/A	N/A	N/A
COPPER	0.006	0.006	0.005	N/A	N/A	N/A
CADMIUM	<0.001	<0.001	<0.001	N/A	N/A	N/A
ZINC	<0.001	<0.001	<0.001	N/A	N/A	N/A
CHROMIUM	0.005	<0.001	<0.001	N/A	N/A	N/A
VANADIUM	0.005	<0.002	<0.002	N/A	N/A	N/A
ALUMINUM	0.264	0.218	0.166	N/A	N/A	N/A
ARSENIC	0.0003	0.0003	0.0003	N/A	N/A	N/A
COBALT	<0.001	<0.001	0.025	N/A	N/A	N/A
NICKEL	0.005	0.003	0.015	N/A	N/A	N/A
LEAD	<0.003	<0.003	<0.003	N/A	N/A	N/A
MANGANESE	<0.008	<0.008	<0.008	N/A	N/A	N/A
BERYLLIUM	<0.001	<0.001	<0.001	N/A	N/A	N/A
MOLYBDENUM	<0.001	<0.001	<0.001	N/A	N/A	N/A
BARIUM	0.024	0.025	0.019	N/A	N/A	N/A
BORON	0.01	0.008	0.013	N/A	N/A	N/A
TN (PARTIC)	N/A	<0.01	0.03	N/A	<0.01	<0.01
OTHERS	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
C.O.D.	N/A	N/A	N/A	N/A	N/A	22.8
AMMONIA	N/A	N/A	N/A	N/A	N/A	0.78
NITROGEN T.K.	N/A	N/A	N/A	N/A	N/A	1.45

LOCATION	< 1986	Edmonton	E.L.Smith	W.T.P. >	E.L. Smith W.T.P.	18110 - 98 Ave., Edm.
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	3-Mar-86
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	Trace	<0.1	<0.1	<0.1	Trace	Trace
Bromodichloromethane	Trace	Trace	<5.0	<5.0	Trace	Trace
Bromoform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	2.5	1.9	2.7	3.4	2.2	2.5
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Methylene chloride	<10	<10	<10	<10	<10	<10
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	Trace	<0.1	0.4	<0.1	Trace	Trace
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	Trace	<0.5
o-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
OTHER COMPOUNDS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
C_4H_{10}	N/A	N/A	N/A	N/A	1 - 5	N/A
C_5H_{12}	N/A	N/A	N/A	N/A	1 - 5	1 - 5
C_6H_{14}	N/A	N/A	N/A	N/A	1 - 5	1 - 5
0052o	N/A	N/A	N/A	N/A	1 - 5	N/A
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	N/A	N/A	<0.10	<0.10	N/A	N/A
Aroclor 1221	N/A	N/A	<0.20	<0.20	N/A	N/A
Aroclor 1232	N/A	N/A	<0.10	<0.10	N/A	N/A
Aroclor 1242	N/A	N/A	<0.05	<0.05	N/A	N/A
Aroclor 1248	N/A	N/A	<0.05	<0.05	N/A	N/A
Aroclor 1254	N/A	N/A	<0.05	<0.05	N/A	N/A
Aroclor 1260	N/A	N/A	<0.10	<0.10	N/A	N/A
Aroclor 1262	N/A	N/A	<0.10	<0.10	N/A	N/A
Aroclor 1268	N/A	N/A	<0.05	<0.05	N/A	N/A

LOCATION	< 1986	Edmonton	E.L.Smith	W.T.P. >	E.L. Smith W.T.P.	18110 - 98 Ave., Edm.
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	3-Mar-86
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS						
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	49	14
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
POLYNUCLEAR AROMATICS						
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	Trace	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

LOCATION	< 1986	Edmonton	E.L.Smith	W.T.P. >	E.L. Smith W.T.P.	18110 - 98 Ave., Edm.
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	3-Mar-86
CHLOROORGANICS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0	<1.0	Trace	<1.0	Trace
diethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	1.8	2	Trace	Trace	1.8	1.4
OTHERCOMPOUNDS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzeneacetaldehyde	N/A	N/A	N/A	N/A	1 - 5	10 - 50
dichloromethyl benzene	N/A	N/A	N/A	N/A	1 - 5	0.5 - 1
8H7N substituted benzene, mol. wt = 11	N/A	N/A	N/A	N/A	0.5 - 1	0.5 - 1
cyclohexanecarboxylic acid	N/A	N/A	N/A	N/A	0.5 - 1	N/A
benzeneacetic acid	N/A	N/A	N/A	N/A	0.5 - 1	0.5 - 1
acetic acid, 2-methylphenyl ester	N/A	N/A	N/A	N/A	0.5 - 1	N/A

Alberta Environment
Treated Water Survey 1986

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
pH	8.2	8.1	8.1	7.6	8.3	8.1
	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L
IRON	<0.02	0.17	<0.02	<0.02	<0.02	0.17
CALCIUM	26	26	27	27	37	26
HARDNESS T	122	114	113	113	129	114
POTASSIUM	0.8	0.7	2.4	0.9	4.9	0.07
NO2+NO3	0.1	0.1	0.11	0.07	0.2	0.1
FLUORIDE	0.95	0.97	1.01	1.15	1	0.97
SULFATE	62	56	64	58	67	56
ALKALINITY	68	69	61	52	67	69
TC (PARTIC)	N/A	0.13	0.45	0.04	0.2	0.13
TURBIDITY	N/A	<0.1	0.2	<0.1	N/A	<0.1
CONDUCTIVITY	268	266	272	250	317	266
TDS	155	145	152	135	178	145
MAGNESIUM	14	12	11	11	9	12
SODIUM	7	4	6	4	9	4
SILICA	4.5	4.2	3.2	3.5	3.9	4.2
NITRITE	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05
CHLORIDE	4	4	5	3	10	4
BICARBONATE	82	85	74	63	82	85
DOC	1.1	0.9	2.1	0.6	<0.4	0.9
DIC	13.8	17.4	12.7	12.1	<1	17.4
MERCURY	<0.0001	<0.0001	<0.0001	<0.0001	N/A	<0.0001
SELENIUM	<0.0002	<0.0002	<0.0002	<0.0002	N/A	<0.0002
COPPER	0.005	0.011	0.007	0.007	N/A	0.011
CADMIUM	<0.001	<0.001	<0.001	<0.001	N/A	<0.001
ZINC	0.002	0.008	0.005	0.004	N/A	0.008
CHROMIUM	0.004	<0.001	<0.001	<0.001	N/A	<0.001
VANADIUM	0.004	0.003	<0.002	<0.002	N/A	0.003
ALUMINUM	0.322	0.204	0.388	0.157	N/A	0.204
ARSENIC	<0.0002	0.0003	0.0004	<0.0002	N/A	0.003
COBALT	<0.001	<0.001	0.026	<0.001	N/A	<0.001
NICKEL	0.004	0.006	0.016	0.003	N/A	0.006
LEAD	<0.003	<0.003	<0.003	<0.003	N/A	<0.003
MANGANESE	<0.008	<0.008	<0.008	<0.008	N/A	<0.008
BERYLLIUM	<0.001	<0.001	<0.001	<0.001	N/A	<0.001
MOLYBDENUM	0.004	<0.001	<0.001	<0.001	N/A	<0.001
BARIUM	0.026	0.029	0.023	0.028	N/A	0.029
BORON	0.022	0.017	0.011	<0.002	N/A	0.017
TN (PARTIC)	N/A	<0.01	<0.01	<0.01	0.06	<0.01

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
CHLORINATED PESTICIDES	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aldrin	<0.500	<0.500	<0.500	<0.500	N/A	N/A
a-Chlordane	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Y-Chlordane	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Captan	<0.050	<0.050	<0.050	<0.050	N/A	N/A
Dacthal	<0.010	<0.010	<0.010	<0.010	N/A	N/A
o,p'-DDD	<0.010	<0.010	<0.010	<0.010	N/A	N/A
p,p'-DDD	<0.010	<0.010	<0.010	<0.010	N/A	N/A
o,p'-DDE	<0.010	<0.010	<0.010	<0.010	N/A	N/A
p,p'-DDE	<0.010	<0.010	<0.010	<0.010	N/A	N/A
o,p'-DDT	<0.020	<0.020	<0.020	<0.020	N/A	N/A
p,p'-DDT	<0.020	<0.020	<0.020	<0.800	N/A	N/A
Diallate (Avadex)	<0.800	<0.800	<0.800	<0.010	N/A	N/A
Dieldrin	<0.010	<0.010	<0.010	<0.040	N/A	N/A
Dursban	<0.040	<0.040	<0.040	<0.010	N/A	N/A
a-Endosulfan	<0.010	<0.010	<0.010	<0.010	N/A	N/A
B-Endosulfan	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Endrin	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Heptachlor	<0.020	<0.020	<0.020	<0.020	N/A	N/A
Heptachlor epoxide	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Hexachlorobenzene	<0.030	<0.030	<0.030	<0.030	N/A	N/A
a-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	N/A	N/A
B-Hexachlorocyclohexane	<0.040	<0.040	<0.040	<0.040	N/A	N/A
Y-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	N/A	N/A
g-Hexachlorocyclohexane	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Kelthane (Dicofol)	<0.030	<0.030	<0.030	<0.030	N/A	N/A
Methoxychlor	<0.030	<0.030	<0.030	<0.030	N/A	N/A
Mirex	<0.010	<0.010	<0.010	<0.010	N/A	N/A
Perthane	<0.300	<0.300	<0.300	<0.300	N/A	N/A
Ramrod (Propachlor)	<0.200	<0.200	<0.200	<0.200	N/A	N/A
Triallate (avadex BW)	<0.040	<0.040	<0.040	<0.040	N/A	N/A
POLYCHLORINATED BIPHENYLS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aroclor 1016	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Aroclor 1221	<0.20	<0.20	<0.20	<0.20	N/A	N/A
Aroclor 1232	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Aroclor 1242	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Aroclor 1248	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Aroclor 1254	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Aroclor 1260	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Aroclor 1262	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Aroclor 1268	<0.05	<0.05	<0.05	<0.05	N/A	N/A

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
ORGANOPHOSPHORUS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Aspon	<0.50	<0.50	<0.50	<0.50	N/A	N/A
Crufomate	<2.00	<2.00	<2.00	<2.00	N/A	N/A
Dasanit	<4.00	<4.00	<4.00	<4.00	N/A	N/A
DDVP	<0.20	<0.20	<0.20	<0.20	N/A	N/A
DEF	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Demeton	<7.00	<7.00	<7.00	<7.00	N/A	N/A
Dimethoate	<0.15	<0.15	<0.15	<0.15	N/A	N/A
Di-Syston	<25.00	<25.00	<25.00	<25.00	N/A	N/A
Ethion	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Fenthion	<1.25	<1.25	<1.25	<1.25	N/A	N/A
Fenitrothion	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Fonofos	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Guthian	<2.00	<2.00	<2.00	<2.00	N/A	N/A
Malathion	<0.17	<0.17	<0.17	<0.17	N/A	N/A
Methyl Parathion	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Methyl Trithion	<0.25	<0.25	<0.25	<0.25	N/A	N/A
Mevinphos	<0.20	<0.20	<0.20	<0.20	N/A	N/A
Mocap	<0.15	<0.15	<0.15	<0.15	N/A	N/A
Paraoxon	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Parathion	<0.06	<0.06	<0.06	<0.06	N/A	N/A
Phorate	<0.20	<0.20	<0.20	<0.20	N/A	N/A
Ronnel	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Tetrachlorvinphos	<0.03	<0.03	<0.03	<0.03	N/A	N/A
ORGANONITROGEN	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Atrazine	<1.00	<1.00	<1.00	<1.00	N/A	N/A
Aminocarb	<100.00	<100.00	<100.00	<100.00	N/A	N/A
Bromacil	<0.05	<0.05	<0.05	<0.05	N/A	N/A
Cobex	<0.03	<0.03	<0.03	<0.03	N/A	N/A
Cyanazine	<0.03	<0.03	<0.03	<0.03	N/A	N/A
Diazinon	<0.50	<0.50	<0.50	<0.50	N/A	N/A
Dinoseb	<0.15	<0.15	<0.15	<0.15	N/A	N/A
Metribuzin	<0.03	<0.03	<0.03	<0.03	N/A	N/A
Prometryne	<1.50	<1.50	<1.50	<1.50	N/A	N/A
Propanil	<0.70	<0.70	<0.70	<0.70	N/A	N/A
Propazine	<1.00	<1.00	<1.00	<1.00	N/A	N/A
Simazine	<1.00	<1.00	<1.00	<1.00	N/A	N/A
Terbacil	<0.10	<0.10	<0.10	<0.10	N/A	N/A
Trifluralin (Treflan)	<0.03	<0.03	<0.03	<0.03	N/A	N/A

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
PURGEABLE PRIORITY POLLUTANTS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzene	Trace	<0.1	<0.1	<0.1	Trace	<0.1
Bromodichloromethane	Trace	Trace	<5.0	<5.0	Trace	Trace
Bromoform	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Carbon Tetrachloride	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chlorobenzene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Chloroform	2.5	1.9	2.7	3.4	3.5	4.2
Dibromochloromethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 2-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 3-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 4-Dichlorobenzene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 1-Dichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one, 2-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one, 1-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,2-Dichloroethylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
one, 2-Dichloropropane	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
cis-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
trans-1,3-Dichloropropylene	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Ethyl benzene	<0.1	<0.1	<0.1	<0.1	<0.1	Trace
Methylene chloride	<10	<10	<10	<10	<10	Trace
one,1,2,2-Tetrachloroethane	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
Tetrachloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Toluene	Trace	<0.1	0.4	<0.1	<0.1	Trace
one,1,1-Trichloroethane	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
one,1,2-Trichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethylene	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2
Trichlorofluoromethane	<0.5	<0.5	<0.5	<0.5	<0.2	<0.5
o-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
m-Xylene	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
p-Xylene	<0.1	<0.1	<0.1	<0.1	0.2	<0.1
OTHERCOMPOUNDS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
C4H10	N/A	N/A	N/A	N/A	1 - 5	1 - 5
C5H12	N/A	N/A	N/A	N/A	1 - 5	1 - 5
C6H14	N/A	N/A	N/A	N/A	1 - 5	1 - 5
0052o	N/A	N/A	N/A	N/A	1 - 5	1 - 5

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
EXTRACTABLE PRIORITY POLLUTANT	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
ACID GROUPS						
Benzoic Acid	<1.0	<1.0	<1.0	<1.0	16	8.9
four-Chloro-m-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dimethylphenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four,6-Dinitro-o-cresol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexadecanoic acid	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Nitrophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pentchlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,5-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4,6-Trichlorophenol	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
BASE NEUTRAL GROUP						ug/L
POLYNUCLEAR AROMATICS	ug/L	ug/L	ug/L	ug/L	ug/L	
Acenaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Acenaphthylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benz (a) anthracene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (k) fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (ghi) perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Benzo (a) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Chrysene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dibenz (ah) anthracene	<1.0	<1.0	Trace	<1.0	<1.0	<1.0
Fluoranthene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Fluorene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Indenol (1,2,3-cd) pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Naphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Perylene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Phenanthrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Pyrene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0

LOCATION	< 1986	Edmonton	Rossdale	W.T.P. >	Rossdale W.T.P.	Oxbridge Place
SAMPLING CLASSIFICATION	Normal Survey	Normal Survey	Normal Survey	Normal Survey	Special Request	Special Request
DATE	JANUARY	FEBRUARY	MARCH	APRIL	3-Mar-86	5-Mar-86
CHLOROORGANICS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Chlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-Chloronaphthalene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,3-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,4-Dichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorobutadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachlorocyclopentadiene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Hexachloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2,4-Trichlorobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
NITROAROMATICS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
three,3'-Dichlorobenzidine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,4-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two,6-Dinitrotoluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
one,2-Diphenylhydrazine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Nitrobenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodimethylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodiphenylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
N-Nitrosodi-n-propylamine	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
HALOESTERS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
four-Bromophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethoxy)methane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroethyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-chloroisopropyl)ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
two-chloroethyl vinyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
four-Chlorophenyl phenyl ether	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
PHTHALATE ESTERS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Butylbenzlyphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-butylphthalate	<1.0	<1.0	<1.0	Trace	Trace	<1.0
diethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Dimethylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Di-n-octylphthalate	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	1.8	2	Trace	Trace	1.3	1.3
OTHERCOMPOUNDS	ug/L	ug/L	ug/L	ug/L	ug/L	ug/L
Benzeneacetaldehyde	N/A	N/A	N/A	N/A	1 - 5	0.5 - 1
H7N substituted benzene, mol. wt = 1	N/A	N/A	N/A	N/A	0.5 - 1	0.5 - 1
Cl2 dichloromethyl benzene, mol. wt.	N/A	N/A	N/A	N/A	0.5 - 1	N/A
C10H14O isomer, mol. wt. = 150	N/A	N/A	N/A	N/A	N/A	0.5 - 1

Appendix F

Enviro-Test Laboratories GC/MS Analytical Results for Spring 1985

7.

ETL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 - 67th Avenue — Edmonton, Alberta T6E 0P5 Telephone: (403) 434-9509

CHEMICAL ANALYSIS REPORT

CITY OF EDMONTON
WATER AND SANITATION
12220 - STONY PLAIN ROAD
EDMONTON, ALTA

DATE: March 25, 1985

ATTN: INNI RASHID

LAB SAMPLE #: 2-85-1271 SAMPLED BY : City

CUSTOMER #: ______ DATE RECEIVED: Feb. 28, 1985

SAMPLE DESCRIPTION: Two water samples for total hydrocarbons and GC/MS identification of the major components.

OVERVIEW:

The samples were extracted for total hydrocarbons and analyzed by GC/FID. The major components present were identified by GC/MS.

RESULTS:

Sample 85-50 - Rossdale North Flume H_2O treated, Feb. 28/85, Sample #2-85-1271-1✓

1. Total hydrocarbons extracted - 93 ug/l
2. GC/MS characterization

COMPOUND IDENTIFIED	APPROX. RELATIVE PERCENTAGE AMOUNT
1. Trimethyl benzenes	5%
2. Tetramethyl benzenes	4%
3. Naphthalene	2%
4. Methyl Naphthalene(two isomers)	15%
5. Dimethyl Naphthalene(three isomers)	13%
6. Trimethyl Naphthalenes(three isomers)	13%
7. Anthracene/phenanthrene	1%

...2/

Sample 85-51 #3 Plant, Feb. 28/85 Sample #2-85-1271-2 ,

1. Total hydrocarbons extracted - 180 ug/l, calculated against a diesel standard.

2. GC/MS characterization

COMPOUND IDENTIFIED	APPROX. RELATIVE PERCENTAGE AMOUNT
1. Trimethyl benzenes	5%
2. Tetramethyl benzenes	4%
3. Naphthalene	2%
4. Methyl Naphthalene(two isomers)	15%
5. Dimethyl Naphthalene(three isomers)	13%
6. Trimethyl Naphthalenes(three isomers)	13%
7. Anthracene/phenanthrene	1%

COMMENTS

1. Other aromatics are present and can be identified by further reduction in final volume of the extracts and re-injection into the Mass spectrometer. The two GC/FID chromatograms Figure 1 and Figure 2 which represent the two samples are identical, thus the composition of the extracts are the same.

CERTIFIED BY ....

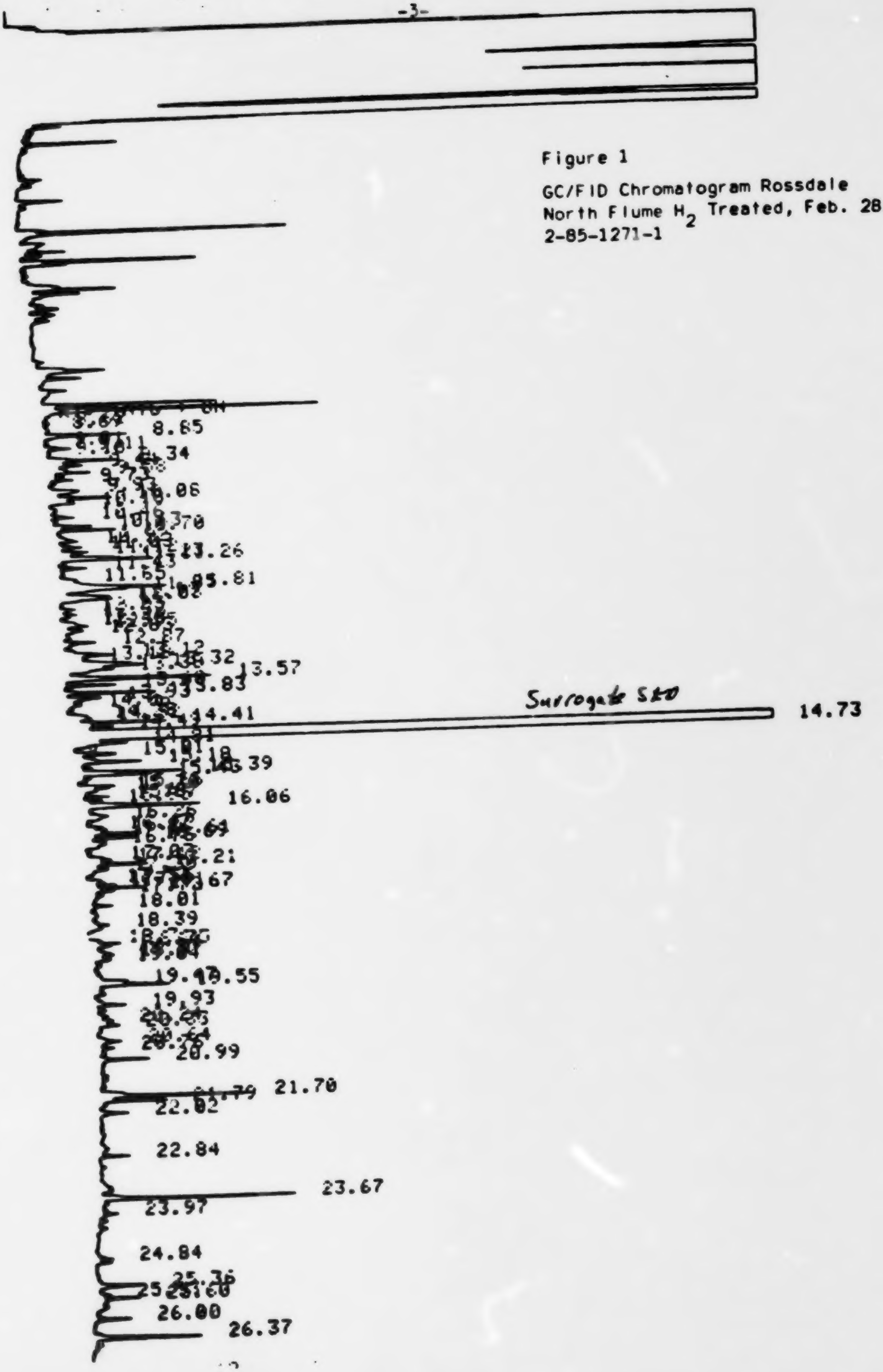


Figure 1

GC/FID Chromatogram Rossdale
North Flume H_2 Treated, Feb. 28/85
2-85-1271-1

Surrogate Std

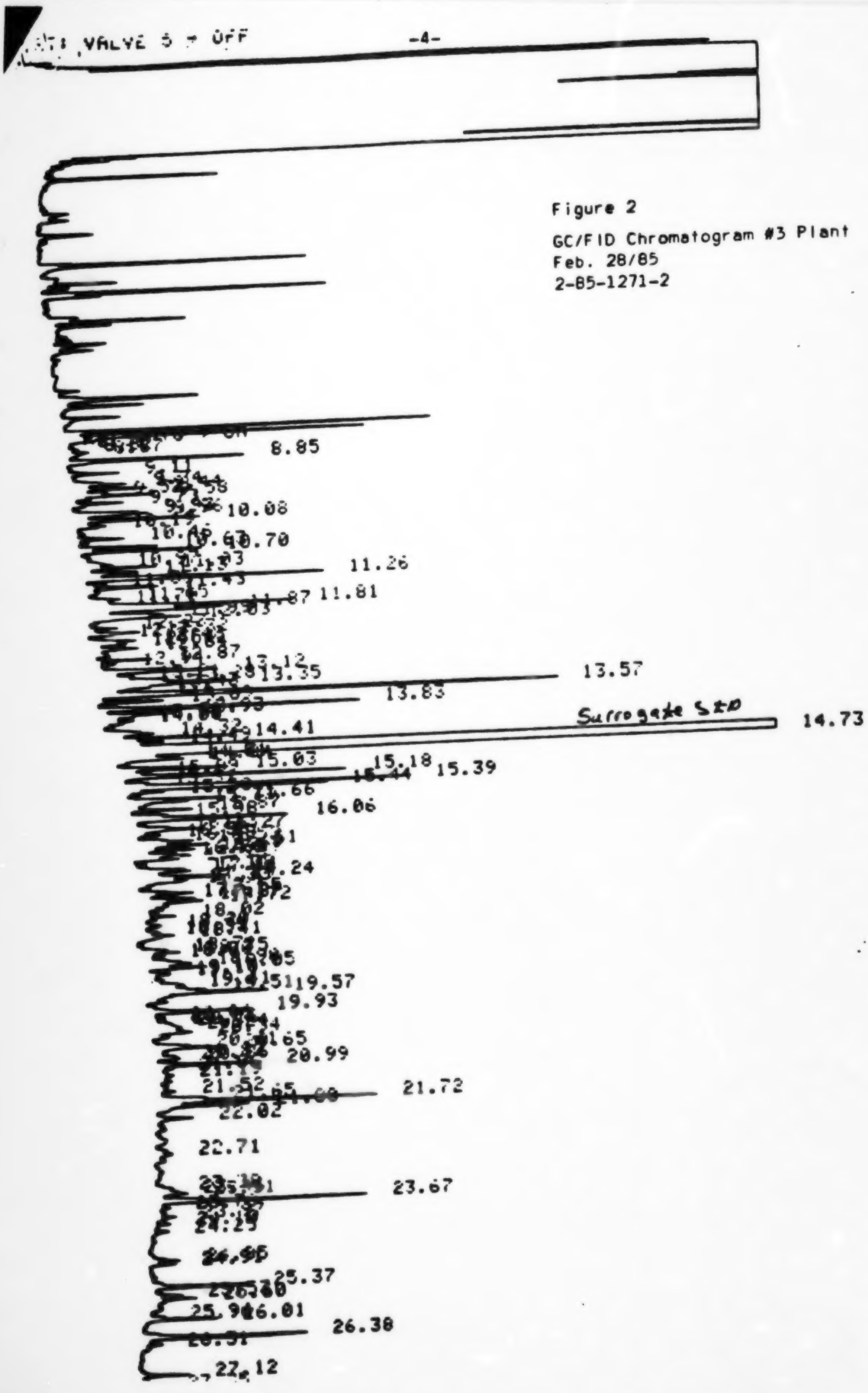


Figure 2

GC/FID Chromatogram #3 Plant
Feb. 28/85
2-85-1271-2

DB 5
{ BONDED CARBOWAX
{ cap column

ETL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 - 67th Avenue — Edmonton, Alberta T6E 0P5 Telephone: (403) 434-9509

CHEMICAL ANALYSIS REPORT

CITY OF EDMONTON
WATER AND SANITATION
12220 - STONY PLAIN ROAD
EDMOTNON, ALTA ATTN: INNI RASHID

DATE: March 25, 1985

LAB SAMPLE #: 3-85-1296 SAMPLED BY :

CUSTOMER #: DATE RECEIVED: March 21/85

SAMPLE DESCRIPTION: 1 water samples for the EPA extraction and analysis of acid fraction. EL Smith potable water, March 21, 1985, 5:00 pm.

---

1) Total Hydrocarbons in the Acid fraction - 12 ug/l (12ppb)

2) Several peaks were present on GC/FID. The sample extract was reduced to 300 uL and analyzed by GC/MS.

COMPOUND IDENTIFIED	CONFIDENCE OF IDENTIFICATION	APPROX. RELATIVE PRECENTAGE AMOUNTS
1. Butyric Acid	2	25%
2. Cyclohexanecarboxylic Acid	2	25%
3. Benzoic Acid	1	25%
4. Benzene~~acidic~~ acetic Acid	2	25%

CERTIFIED BY ....

TABLE OF CONFIDENCE OF IDENTIFICATION OF GC/MS SPECTRA

Number 1: Very confident, clean spectra, excellent match with library spectra.

Number 2: Some small spectra differences with library match. A good comparison. Reference standard needed for positive identification.

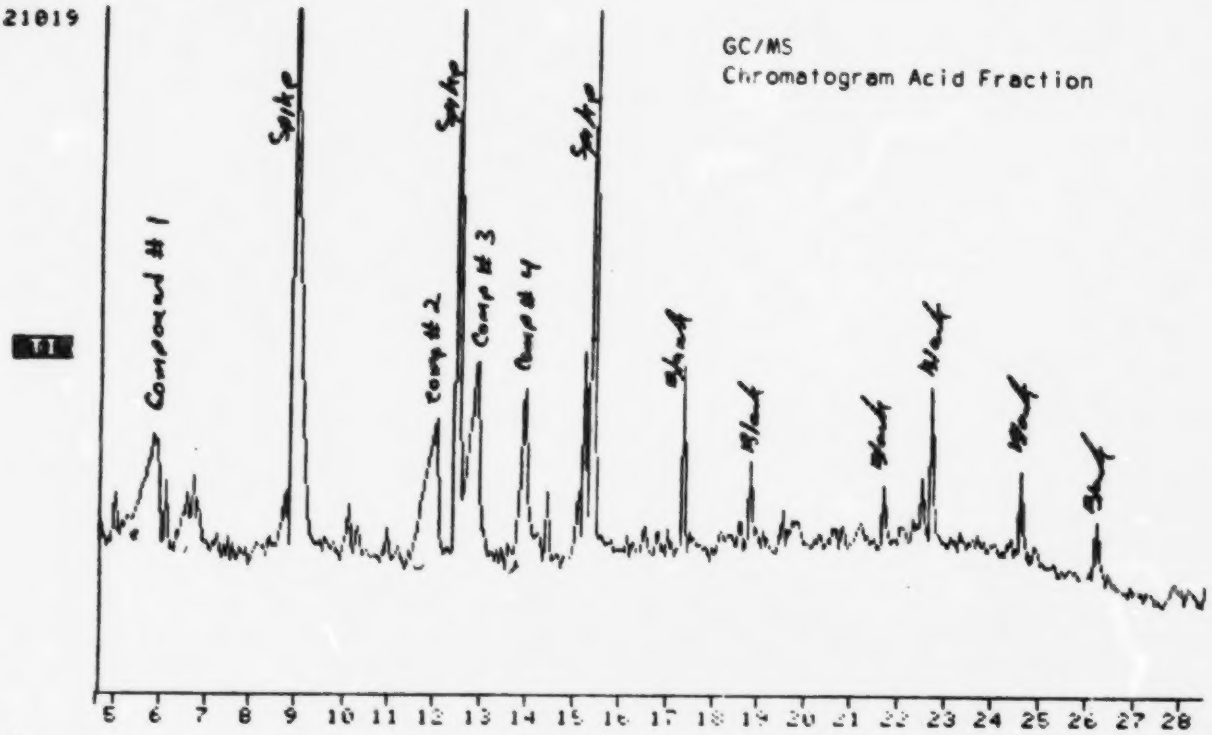
Number 3: Possible compound or very similar to. Spectra is difinitely contaminated. Reference standard needed for positive identification.

Number 4: Evidence of possible structure and molecular weight. Compound not identified.

Number 5: Unable to identify.

NAME :1296-ACID,3UL,DE
MISC :1800V,4004-300010 MIN,C15,22/3 85

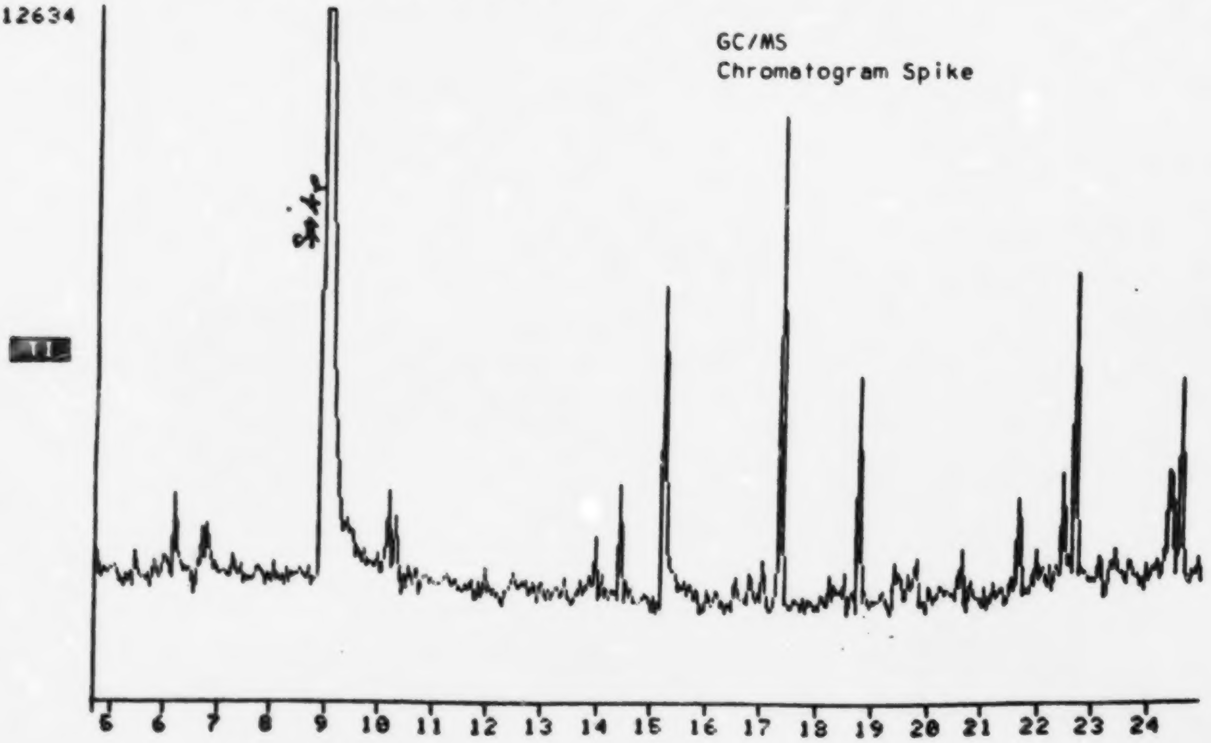
FRN 15004



GC/MS
Chromatogram Acid Fraction

NAME 1296-ACID BLANK,3UL,DE
MISC 1800V,40/4-300010/MIN,22/3/85,C15

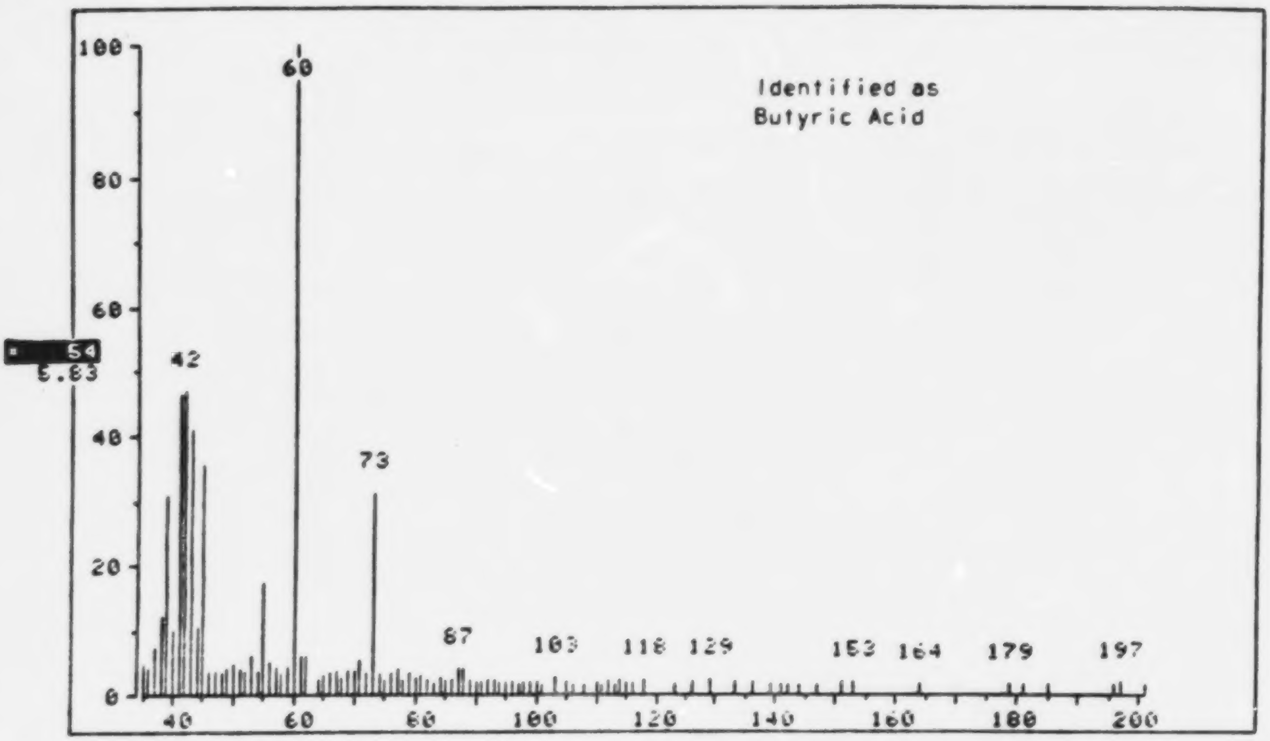
FRN 15005



GC/MS
Chromatogram Spike

1296-ACID,3UL,DE
1800V,4004-300010/MIN,C15,22/3/85

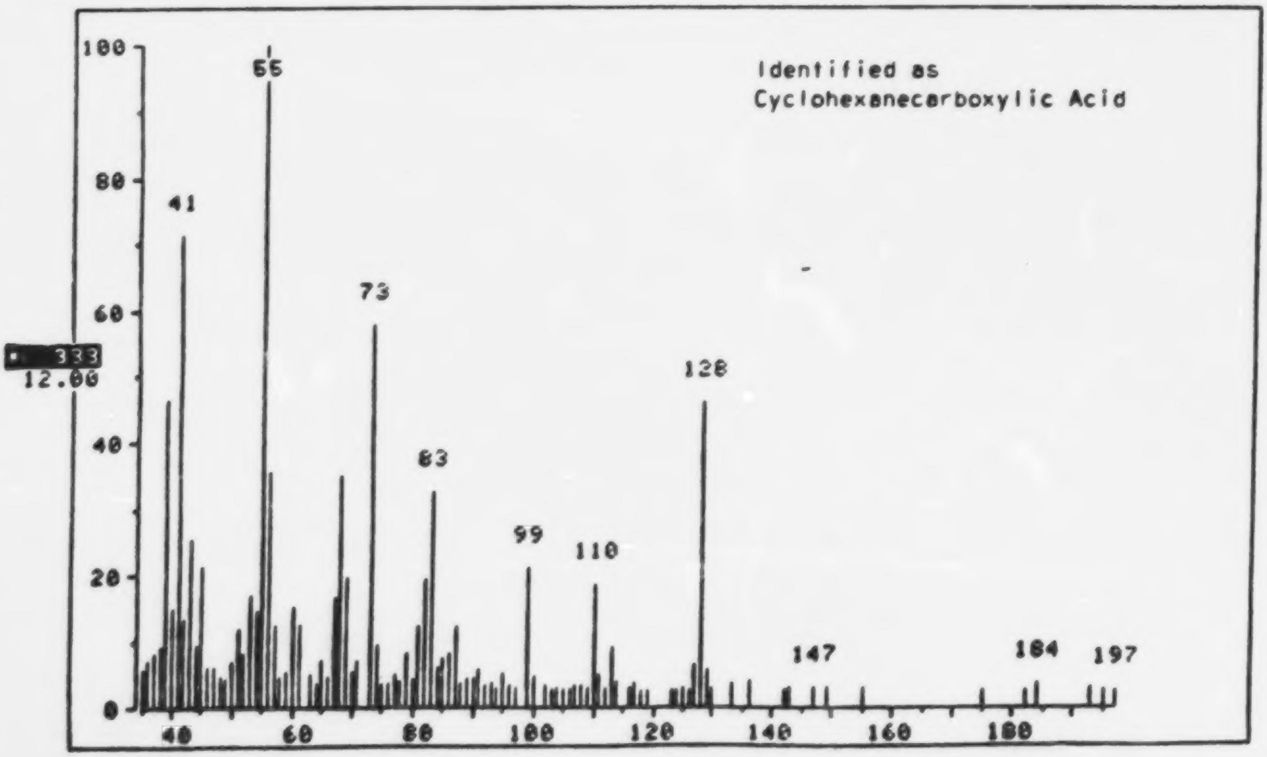
[illegible] 15004



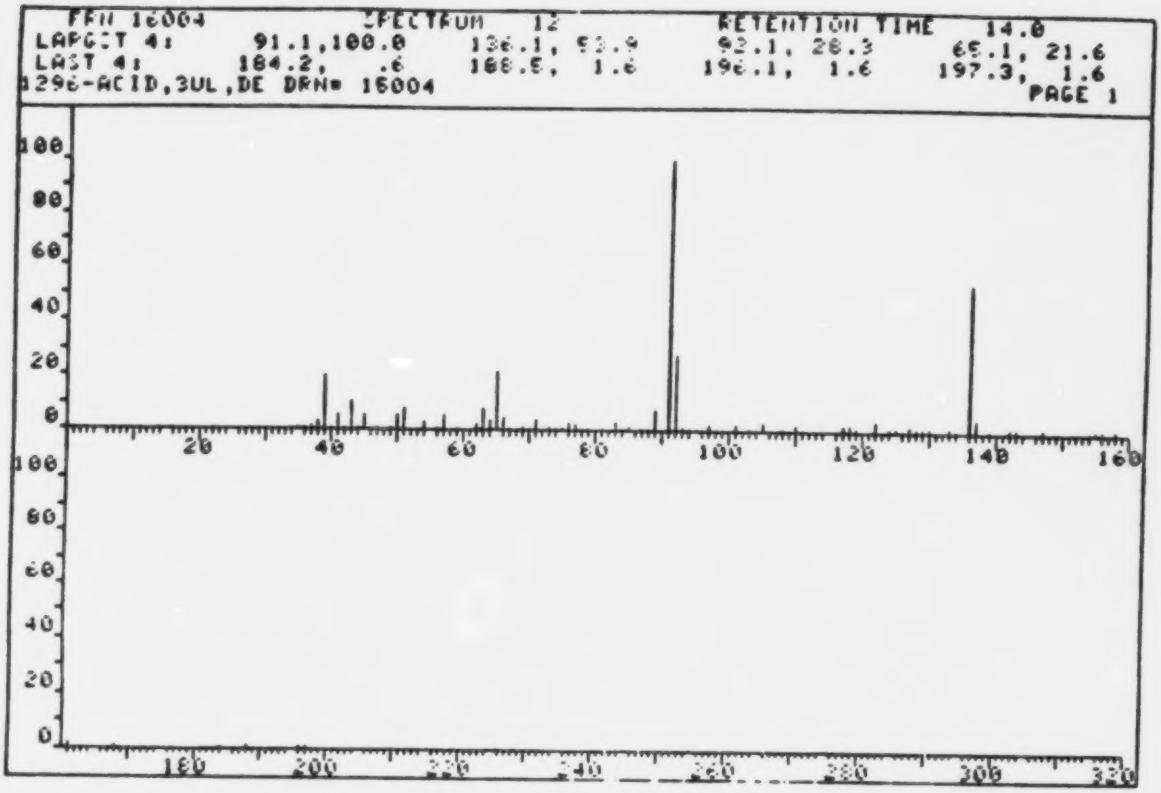
Identified as Butyric Acid

1296-ACID,3UL,DE
1800V,4004-300010/MIN,C15,22/3/85

FPN 15004

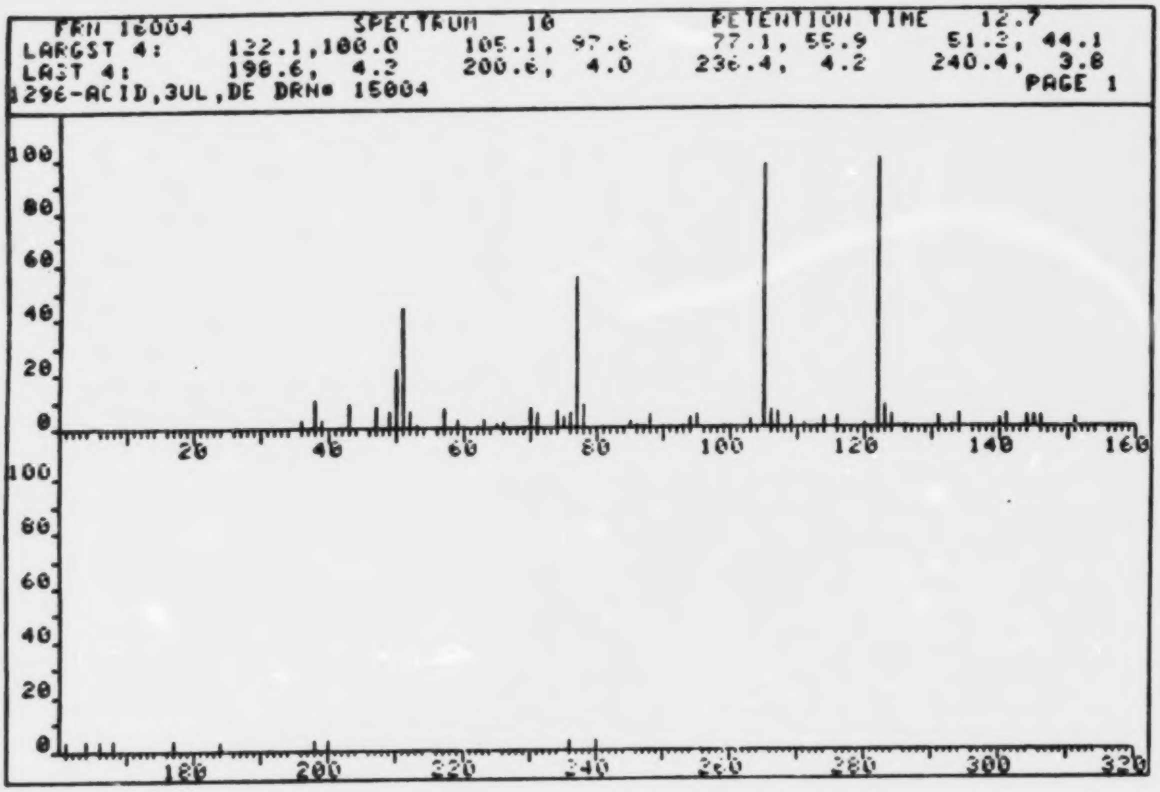


Identified as Cyclohexanecarboxylic Acid



```
  FRN 16004            SPECTRUM    12            RETENTION TIME    14.0
LARGEST 4:      91.1,100.0    136.1, 53.9     92.1, 28.3      65.1, 21.6
LAST 4:        184.2,    .6   188.5,  1.6    196.1,  1.6     197.3,  1.6
1296-ACID,3UL,DE DRN= 16004                                      PAGE 1
```

Identified as
Benzeneacetic Acid



```
  FRN 16004            SPECTRUM   10            RETENTION TIME   12.7
LARGST 4:    122.1,100.0    105.1, 97.6     77.1, 55.9      51.2, 44.1
LAST 4:      198.6,  4.2    200.6,  4.0    236.4,  4.2     240.4,  3.8
1296-ACID,3UL,DE DRN# 15004                                    PAGE 1
```

Identified as
Benzoic Acid

ETL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 - 67th Avenue — Edmonton, Alberta T6E 0P5 **Telephone: (403) 434-9509**

CHEMICAL ANALYSIS REPORT

City of Edmonton
Water & Sanitation
Rossdale Water Treatment Plant

DATE: April 1, 1985

ATTN: Inni Rashid

LAB SAMPLE #: 3-85-1302 SAMPLED BY : City

CUSTOMER #: DATE RECEIVED: March 28, 1985

SAMPLE DESCRIPTION: 1 water sample, March 1, Venture Inn, Hot for Purge & Trap characterization of volatiles.

VOLATILES IDENTIFIED	CONCENTRATION
1. 2-Methyl propanal	Less than 5 ppb, concentration not accurately determined
2. Chloroform	3 ppb
3. 3-Methyl butanal	Less than 5 ppb, concentration not accurately determined
4. 2-Methyl butanal	Less than 5 ppb, concentration not accurately determined
5. Dimethyl benzenes (Isomers) (Xylenes)	Less than 5 ppb, concentration not accurately determined
6. Trimethyl benzenes (Isomers)	Less than 5 ppb, concentration not accurately determined

Certified By....................................

ETL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 - 67th Avenue — Edmonton, Alberta T6E 0P5 Telephone: (403) 434-9509

CHEMICAL ANALYSIS REPORT

CITY OF EDMONTON
WATER AND SANITATION
12220 - STONY PALIN ROAD
EDMONTON, ALTA
T5N 3Y4

DATE: March 25, 1985

ATTN: INNI RASHID

LAB SAMPLE #: 3-85-1296-2

SAMPLED BY : City

CUSTOMER #:

DATE RECEIVED: March 20, 1985

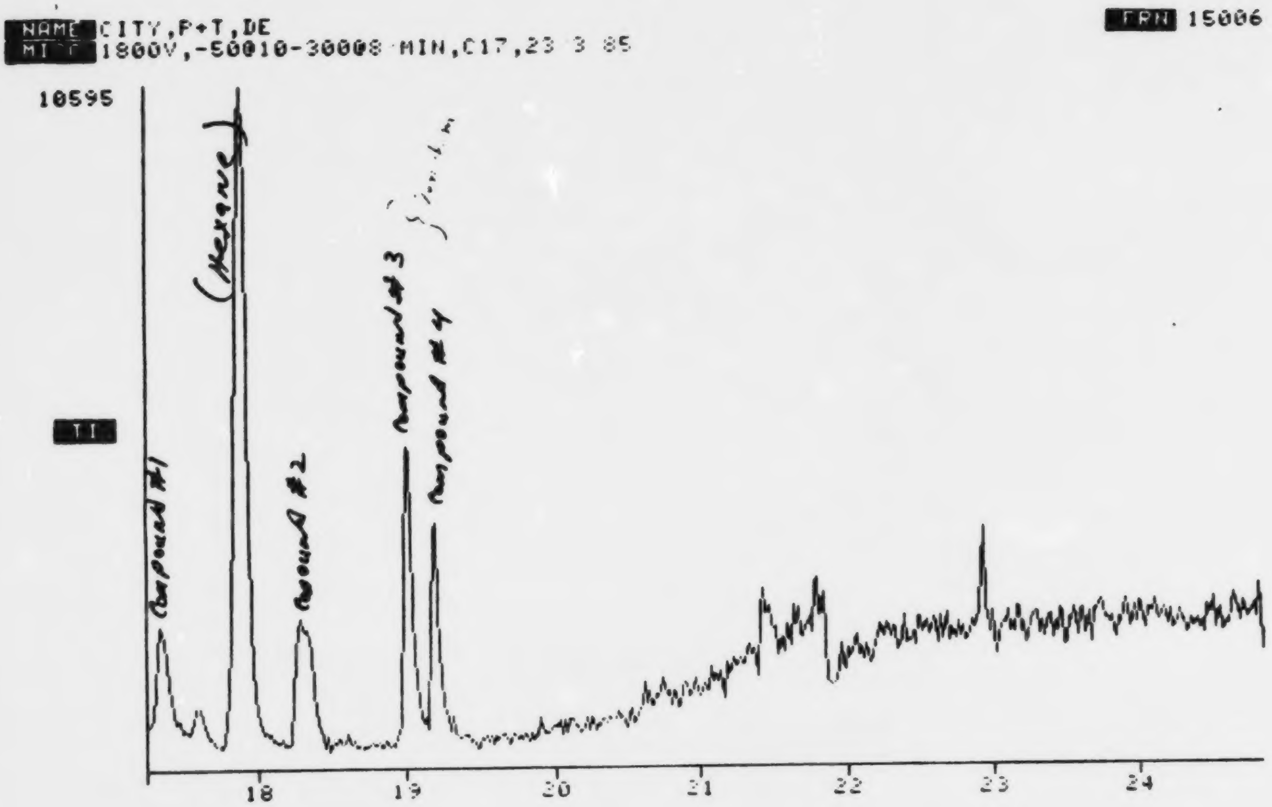
SAMPLE DESCRIPTION: 1 water sample taken from EL Smith Plant morning sample, March 20, 1985 for purge and trap analysis. GC/MS identification.

COMPOUND IDENTIFIED BY PURGE AND TRAP	CONFIDENCE OF IDENTIFCATION
1. 2-methyl propanal	2
2. Chloroform	1
3. 3-methyl butanal	2
4. 2-methyl butanal	2

CERTIFIED BY ....

TABLE OF CONFIDENCE OF IDENTIFICATION OF GC/MS SPECTRA

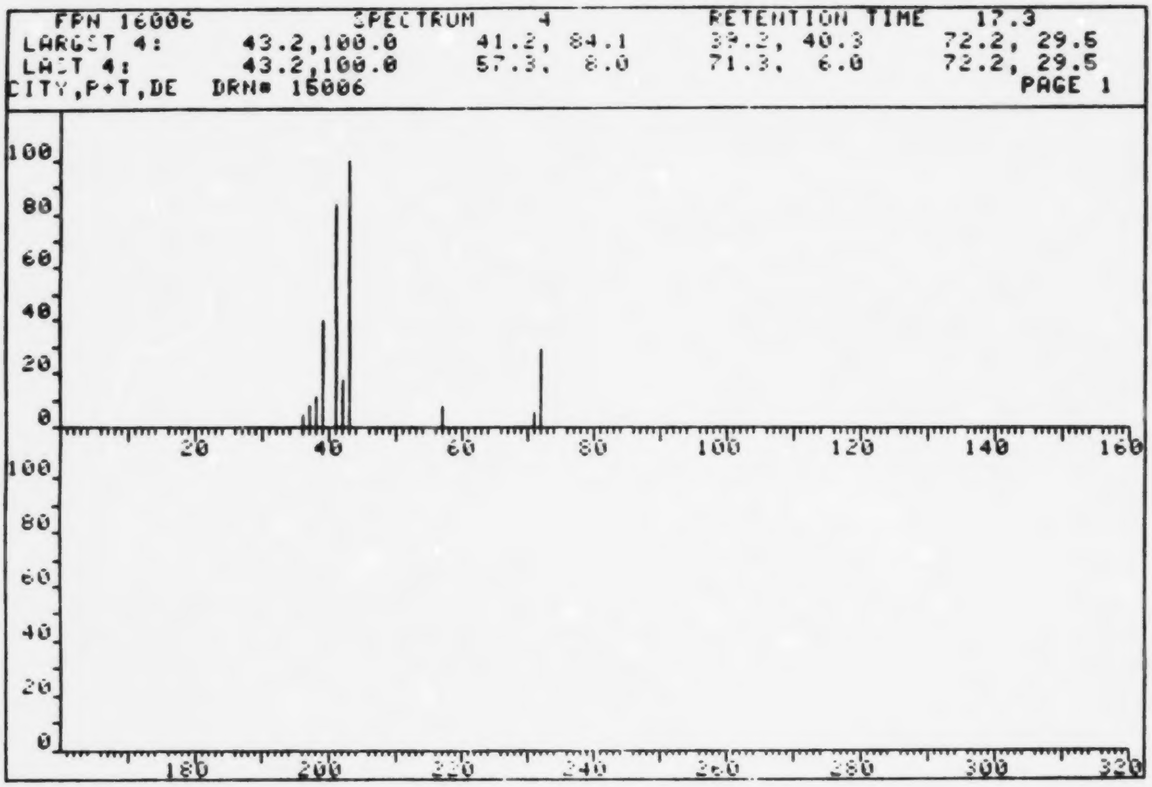
Number 1:	Very confident, clean spectra, excellent match with library spectra.
Number 2:	Some small spectra differences with library match. A good comparison. Reference standard needed for positive identification.
Number 3:	Possible compound or very similar to. Spectra is difinitely contaminated. Reference standard needed for positive identification.
Number 4:	Evidence of possible structure and molecular weight. Compound not identified.
Number 5:	Unable to identify.



GC/MS

Chromatogram of 3-85-1296-2

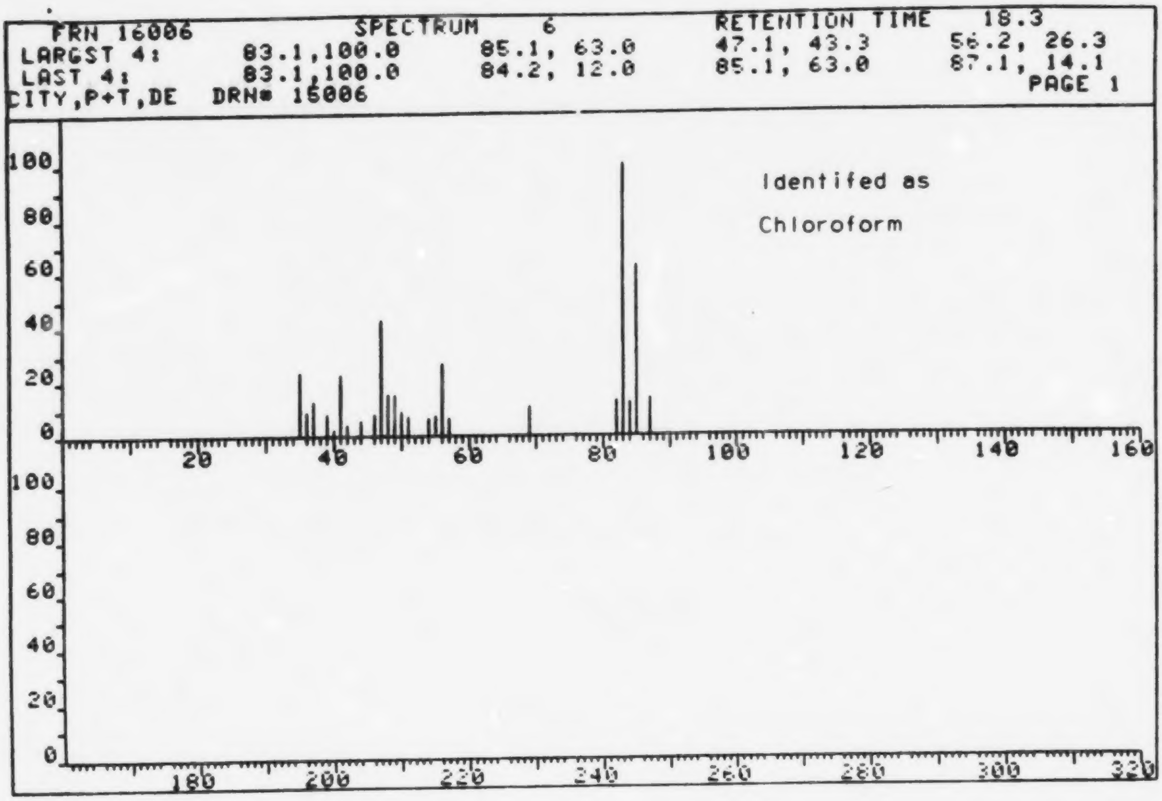
Purge and Trap



```
  FPN 16006            SPECTRUM     4           RETENTION TIME    17.3
 LARGST 4:       43.2,100.0       41.2, 84.1       39.2, 40.3       72.2, 29.5
 LAST 4:         43.2,100.0       57.3,  8.0       71.3,  6.0       72.2, 29.5
CITY,P+T,DE   DRN# 16006                                                PAGE 1
```

Identified as

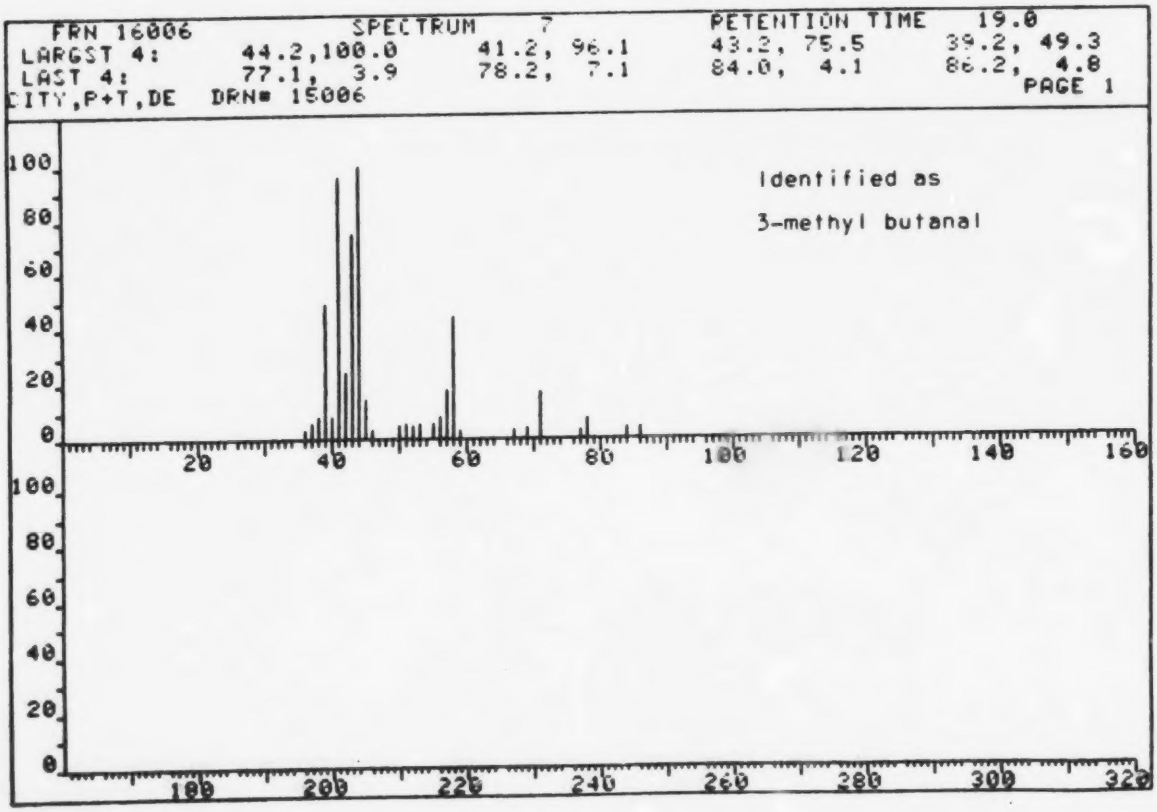
2-methyl propanal



```
  FRN 16006              SPECTRUM    6             RETENTION TIME    18.3
LARGST 4:         83.1,100.0     85.1, 63.0       47.1, 43.3       56.2, 26.3
LAST 4:           83.1,100.0     84.2, 12.0       85.1, 63.0       87.1, 14.1
CITY,P+T,DE   DRN# 15006                                                PAGE 1
```

Identifed as

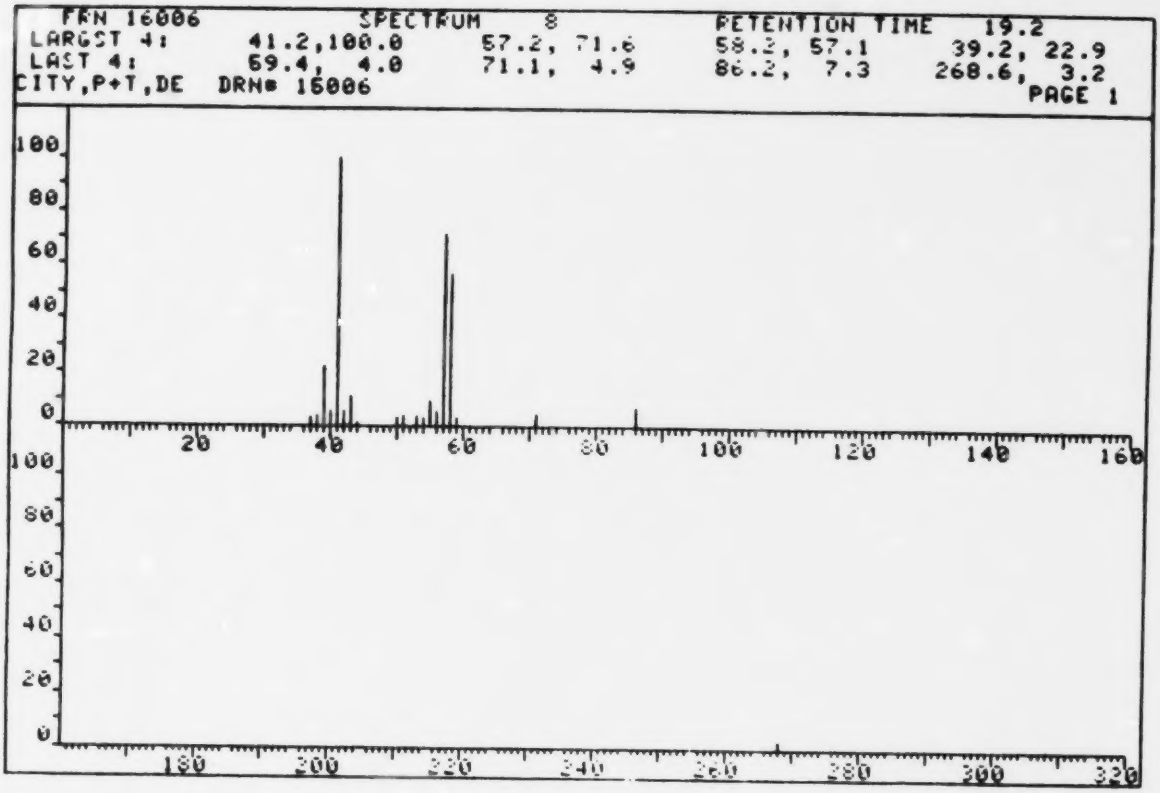
Chloroform



```
  FRN 16006              SPECTRUM    7             RETENTION TIME    19.0
LARGST 4:         44.2,100.0     41.2, 96.1       43.2, 75.5       39.2, 49.3
LAST 4:           77.1,  3.9     78.2,  7.1       84.0,  4.1       86.2,  4.8
CITY,P+T,DE   DRN# 15006                                                PAGE 1
```

Identified as

3-methyl butanal



```
  FRN 16006           SPECTRUM     8            RETENTION TIME    19.2
 LARGST 4:       41.2,100.0      57.2, 71.6     58.2, 57.1        39.2, 22.9
 LAST 4:         69.4,   4.0     71.1,  4.9     86.2,  7.3       268.6,  3.2
CITY,P+T,DE  DRN# 16006                                                PAGE 1
```

Identified as

2-methyl butanal

Appendix G

Specialized Extraction and GC/MS Analyses on 1986 Treated Water Sample by Dr. Coutts

I. SAMPLE RECEIVED

Edmonton water. **(sampled at Brander Gardens, March 5, 1986)**

II. PURIFICATION OF SOLVENTS AND CLEANING OF GLASSWARE

Solvents were glass-distilled prior to use and stored in amber bottles. Caps were lined with aluminum foil.

Glassware was soaked overnight in a solution of Count-Off (New England Nuclear), rinsed with distilled, deionized water (Milli-Q System, Millipore) followed sequentially by ethanol and methylene chloride. Glassware was then heated in a muffle furnace at 400 C for 4 hours to remove traces of organic material.

III. EXTRACTION PROCEDURE

The sample (1 L) was basified to pH 11 (4 drops of a concentrated NaOH solution) and extracted with methylene chloride (3 x 400 mL). The aqueous phase was set aside to be later evaluated for phenols and strong acids. The methylene chloride extracts, containing basics-neutrals, were combined in a 1 L erlenmeyer flask containing sodium sulfate as drying agent. The sample was then transferred to a Kuderna Danish apparatus consisting of a 5 mL graduated concentrator tube, 500 mL flask, and three-ball Snyder column. A boiling chip was added and the apparatus immersed in a hot (85 C) water bath to just below the bottom joint. The solution was concentrated until 1 mL remained in the concentrator tube at which time the apparatus was removed from the bath and allowed to cool. Solvent vapor in the column and flask quickly condensed to give a final volume of 4 - 5 mL in the concentrator tube. The tube was disconnected from the rest of the apparatus and the volume reduced to approximately 1 mL by means of a gentle nitrogen stream and warm (50 C) water bath. The sample was transferred to a 4 mL vial, concentrated to 20 - 50 uL (nitrogen stream), and 1 - 2 uL used for analysis by gas chromatography (GC) and combined GC-mass spectrometry (GC-MS). This is the basics-neutrals fraction.

The aqueous phase was analyzed for the presence of phenols using a previously described aqueous acetylation method [J. Chromatogr., 195, 105(1980); J. Assoc. Off. Anal. Chem., 64, 833(1981); J. Chromatogr. Sci., 19, 151(1981)]. The water was transferred to a 2 L flask and treated with potassium bicarbonate (approximately 30 - 50 g, such that excess bicarbonate remained at the bottom of the flask) and acetic anhydride (5 mL). The flask was shaken vigorously until the evolution of the carbon dioxide had ceased, giving a solution with a final pH of 8. The sample was extracted with methylene chloride (3 x 300 mL) and the combined extracts concentrated and analyzed as described above. This is the phenolic (acetylated) fraction. The aqueous phase was acidified to pH 2 with conc. HCl and extracted with methylene chloride (3 x 300 mL) and the combined extracts concentrated and analyzed as described above. This is the strong acids fraction.

IV. INSTRUMENTATION

Gas Chromatography

Analyses were conducted on a Hewlett Packard gas chromatograph (Model 5700A) equipped with a flame ionization detector (Model 18710A), capillary column control (Model 18740B) and recorder-integrator (Model 3390A). Capillary chromatography was conducted on a 20m x 0.2 mm i.d. fused silica DB 1 (film thickness 0.5 micron) column (J & W Scientific). The operating conditions were: column temperature 50 (2 min) - 270 C (4 C/min) and hold until baseline achieved; detector, 250 C; injector, 250 C; carrier gas (helium) flow rate 1 mL/min.

GC-Mass Spectrometry

Mass spectra were recorded on a VG 7070E spectrometer (VG Analytical) coupled to a Varian Vista 6000 GC and PDP 11 data system. Except for minor changes in the temperature program for some samples, GC operating conditions were identical to those reported above. For GC-MS, a 30m x 0.25 mm i.d. fused silica DB 1 (film thickness 0.25 micron) column (J & W Scientific) was used.

The NBS Library was used for computer matching

V. RESULTS AND DISCUSSION

Each listing includes the mass spectrum number (#) which identifies the component being discussed, the most probable molecular formula (and molecular weight) of the component as determined by computer library match, and our comments based on examination of the spectra from first principles.

Edmonton Water Sample - Acid Fraction

#273 $C_7H_{12}O_2$ (128) cyclohexanecarboxylic acid

Identity - high probability.

#406 $C_7H_6O_2$ (122) benzoic acid

Identity - virtually certain.

#452 $C_8H_8O_2$ (136) benzeneacetic acid OR 3-methylbenzoic acid

Benzeneacetic acid best fit; identity - high probability.

#545 $C_9H_{10}O_2$ (150) benzenepropanoic acid OR formic acid, 2-phenylethyl ester

Propanoic acid better fit since M - HCOOH (104) is base peak for the formate.

Identity - virtually certain.

#854 $C_{13}H_{10}O$ (182) diphenylmethanone

This is benzophenone; identity - virtually certain.

#1081 $C_{14}H_{28}O_2$ (228) tetradecanoic acid

This is myristic acid; identity - medium probability.

#1126 $C_{13}H_{38}$ (254) octadecane

Long chain aliphatic hydrocarbon; identity - virtually certain.

#1141 No matches

Contaminated by component #1152.

#1152 $C_{16}H_{22}O_4$ (278) 1,2-benzenedicarboxylic acid, butyl 2-methylpropyl ester

This is a phthalate; identity - virtually certain.

#1192 $C_{15}H_{30}O_2$ (242) pentadecanoic acid

A good fit for long chain aliphatic acid; identity - medium to high probability.

#1255 $C_{16}H_{22}O_4$ (278) 1,2-benzenedicarboxylic acid, butyl 2-methylpropyl ester

OR dibutyl ester

This is a phthalate; either one possible; identity - virtually certain.

#1312 $C_{16}H_{32}O_2$ (256) hexadecanoic acid

This is palmitic acid; identity - medium to high probability.

#1348 $C_{21}H_{14}N_2O_3Fe$ (398)iron, tricarbonyl N-(phenyl-2-pyridinylmethylene)benzenam

Unlikely; this is a long chain aliphatic hydrocarbon with molecular weight >283.

#1361 No matches

Looks like a long chain aliphatic hydrocarbon.

#1421 $C_{18}H_{36}$ (252) 1-octadecene

Or isomer; long chain unsaturated aliphatic hydrocarbon.

#1511 $C_{18}H_{36}O_2$ octadecanoic acid

This is stearic acid; identity - medium to high probability.

#1552 $C_{22}H_{46}$ (310) docosane

Unlikely; this is a long chain aliphatic hydrocarbon with molecular weight >311.

#1648 $C_{25}H_{52}$ (352) pentacosane

This is a long chain aliphatic hydrocarbon, molecular weight >325.

#1740 $C_{21}H_{14}N_2O_3Fe$(398) iron, tricarbonyl N-(phenyl-2-pyridinylmethylene)benzenam

Unlikely; this is a long chain aliphatic hydrocarbon, molecular weight >339.

#1788 No matches

This is a long chain aliphatic hydrocarbon with aromatic component?

#1836 $C_{24}H_{38}O_4$ (390) 1,2-benzenedicarboxylic acid, diisooctyl ester

Possible; this is a phthalate.

#1850 No matches.

Unable to characterize; only one major fragment m/z 59.

#1914 $C_{26}H_{54}$ (366) hexacosane

Long chain aliphatic hydrocarbon; molecular weight >367.

#1996 $C_{25}H_{52}$ (352) pentacosane

Long chain aliphatic hydrocarbon; molecular weight >381.

#2017 No matches

Virtually identical to #1850; overlap with #2032.

#2032   No matches

Virtually identical to #1850.

#2076 $C_{25}H_{52}$ (352)   pentacosane

Long chain aliphatic hydrocarbon; molecular weight >395.

#2097   No matches

Identity unknown.

#2140 $C_{24}H_{38}O_4$ (390)   1,2-benzenedicarboxylic acid, bis(1-methylheptyl) ester

This is a phthalate.

#2153 $C_{25}H_{52}$ (352)   pentacosane

Long chain aliphatic hydrocarbon; molecular weight >409.

#2276 $C_{27}H_{46}O$ (386)   (3 beta)-cholest-5-en-3-ol

Could be a steroid but one with higher molecular weight.

#2418   No matches

Identity unknown.

Edmonton Water Sample - Base-Neutrals Fraction

#881 $C_{16}H_{30}O_4$ (286)   propanoic acid, 2-methyl-, 2-ethyl-1-propyl-1,3-propanedi

Very unlikely; contains m/z 43 ($C_3H_7^+$ or CH_3CO^+) and m/z 71 ($C_5H_{11}^+$).

I

#943 $C_{15}H_{24}O$ (220) 4-nonylphenol

Good possibility; m/z 107 ($OHPhCH_2^+$) suggests phenol; 191 ($M - C_2H_5$) and 149 ($M - C_5H_{11}$) suggest long aliphatic chain.

#947 $C_{15}H_{24}O$ (220) 4-nonylphenol

Spectrum virtually identical to #943; overlapping peaks.

#954 $C_{15}H_{24}O$ (220) 4-nonylphenol

Similar spectrum to #943 with exception of intense m/z 163 ($M - C_4H_9$) and less intense m/z 177 ($M - C_3H_7$).

#956 $C_{15}H_{24}O$ (220) 4-nonylphenol

Similar to #954; overlapping, or same peak.

#962 $C_{14}H_{22}O$ (206) phenol, 4-(1,1,3,3-tetramethylbutyl)

Unable to characterize; only one significant fragment - base peak m/z 135 suggests $HOPhC^+(CH_3)_2$ which is present in this phenol.

#965 No fits found

Spectrum very similar to #943.

#1010 and #1054 1,2-benzenedicarboxylic acid, butyl 2-methylpropyl ester OR dibutyl ester

Spectra are very similar; appear to be related phthalates.

#1122 $C_{18}H_{36}$ (252) (E)-3-octadecene

Long chain hydrocarbon, possibly unsaturated; an octadecene or lower molecular weight hexadecene.

#1234 No fits found

Unable to characterize; unsufficient fragments.

#1290 $C_{24}H_{38}O_4$ (390) 1,2-benzenedicarboxylic acid, diisooctyl ester

Identity - virtually certain.

#1392 $C_{17}H_{28}O_2$ (264) 2,6,10-dodecatrien-1-ol,3,7,11-trimethyl-, acetate

Insufficient fragments to characterize.

Edmonton Water Sample - Acetylated Fraction

#26 No fits found

Low molecular weight (<150); contains an acetate group; M^+ may be m/z 143; intense m/z 101 (143 - 42); identity unknown, could be an acetylated C_5 cyclic alkanolamine.

#59 $C_5H_8O_2$ (100) 2,4-pentanedione

Identity - high probability.

#71 No fits found

Mixture of #59 and another compound mol. wt. 124 which readily loses a CH_3 group.

#81 $C_{12}H_{24}$ (168) (E)-6-methyl-3-undecene

Identity unknown; mixture of minor components.

#101 C_8H_8O (120) 1-phenylethanone

This is acetophenone; some contamination with higher molecular weight compound; definitely a compound of partial structure PhCOR; identity - high probability

#118 $C_7H_{10}O_3$ (142) 2,4,6-heptanetrione

Identity - medium probability.

#122 C_8H_8O (120) 4-methylbenzaldehyde

Identity - virtually certain.

#132 No fits found

Mixture - ignore.

#143 $C_8H_8O_2$ (136) benzoic acid, methyl ester

Identity - virtually certain.

#154 No fits found

Unidentified minor component.

#195 $C_9H_{16}O_3$ (172) butanoic acid, 2-acetyl-3-methyl-, ethyl ester

Unidentified.

#233 $C_9H_{10}O_2$ (150) acetic acid, phenylmethyl ester

This could be an acetylated methylphenol (cresol); identity - medium to high probability

#251 No fits found

Unidentified.

#278 $C_{10}H_{12}O_2$ (164) benzenemethanol, alpha-methyl-, acetate

Identity - virtually certain.

#306 $C_{12}H_{24}O$ (184) cyclododecanol

Unidentified.

#387 $C_{10}H_{12}O_2$ (164) benzenemethanol, alpha-methyl-, acetate OR phenol, 3-ethyl-, acetate

Isomer of #278; possibly a ring methylated benzyl alcohol (acetylated); identity - medium to high probability

#451 $C_{12}H_{22}$ (166) cyclohexane, (cyclopentylmethyl)

Identity - medium to high probability.

#616 $C_{14}H_{30}$ (198) tetradecane

Alkane, minor branching; identity - virtually certain.

#667 and #671 $C_{14}H_{20}O_2$ (220) 2,6-bis(1,1-dimethylethyl)-2,5-cyclohexadiene-1,4-dione

Single component; this is 2,6-di-tert-butyl-1,4-benzoquinone; identity - high probability

#687 and #699 No fits found

Major peak, but unidentified; partial structure may be $CH_3C_9H_{12}COOC_4H_9$ OR $CH_3C_{11}H_{16}OC_4H_9$.

#716 and #720 No fits found

Mixture of unknown compounds.

#731 and #736 No fits found

Mixture, unidentified.

#742 and #752 $C_{12}H_{18}O$ (178) alpha, alpha-dimethylbenzyl isopropyl ether

Identity - medium to high probability.

#811 $C_{11}H_{12}O_4$ (208) 3,5-diacetyl-2,6-dimethyl-4H-pyran-4-one

Identity - high probability.

#855 $C_{16}H_{30}O_4$ (286) propanoic acid, 2-methyl-, 1-(1,1-dimethylethyl)-2-methyl

Spectrum has the reported base peak of m/z 71 but no other important diagnostic fragments other than m/z 43; isobutyric acid derivative is likely.

#886 $C_{16}H_{34}$ (226) hexadecane

Identity - virtually certain.

#957 $C_{17}H_{18}$ (222) naphthalene, 1,2,3,4-tetrahydro-6-(phenylmethyl)-

Polyunsaturated compound containing a methyl group; probably not correct identity, since the reported base peak m/z 131 is absent; $C_{17}H_{18}$ OR CH_3-$C_{16}H_{15}$ likely.

#981 and #987 $C_{18}H_{20}$ (236) 1H-indene, 2,3-dihydro-1,1,3-trimethyl-3-phenyl-

Polyunsaturated compound; higher homolog of #957; $C_{18}H_{20}$ OR CH_3-$C_{17}H_{17}$; identified compound is a good possibility.

#998 and #1009 $C_{15}H_{24}O$ (220)   4-nonylphenol

Most likely a phenol ether because it is not acetylated; an octyl ether of a cresol is a distinct possibility - (CH_3)-$PhOC_8H_{17}$.

#1042 $C_{14}H_{22}O$ (206)   phenol, 4-(2,2,3,3-tetramethylbutyl)-

Only one major fragment (m/z 135); minor component - ignore.

#1051 $C_{15}H_{24}O$ (220)   nonylphenol OR phenol, 2-methyl-4-(1,1,3,3-tetramethylbutyl)-

Similar to #998 - a cresol ether.

#1087 $C_{14}H_{28}O_2$ (228)   tetradecanoic acid

Possible good match; this is myrisitic acid, a long chained fatty acid.

#1120 $C_{18}H_{20}$ (236)   benzene, 1,1'-(3,3-dimethyl-1-butenylidene)bis- OR 1H-indene, 2,3-dihydro-1,1,3-trimethyl-3-phenyl

Minor component; related in structure to #981; polyunsaturated aromatic compound.

#1130 $C_{18}H_{38}$ (254)   octadecane

Identity - virtually certain.

#1146 $C_{14}H_{28}O_2$ (228)   tetradecanoic acid

Identity unknown.

#1156 $C_{16}H_{22}O_4$ (278)   1,2-benzenedicarboxylic acid, butyl 2-methylpropyl ester

This is a phthalate; only one intense fragment (m/z 149).

#1191   No fits found

Very minor component; polyaromatic compound which contains a nitrogen atom; $C_{18}H_{21}N$ OR CH_3-$C_{17}H_{18}N$ are distinct possibilities.

#1198 $C_{15}H_{30}O_2$ (242) pentadecanoic acid

Very minor component; pentadecanoic acid or a branched isomer is likely.

#1260 $C_{16}H_{22}O_4$ (278) 1,2-benzenedicarboxylic acid, butyl 2-methylpropyl ester OR dibutyl ester

Identity - virtually certain for the butyl methylpropyl ester.

#1283 $C_{16}H_{30}O_2$ (254) 9-hexadecenoic acid

Identity - high probability.

#1300 $C_{12}H_{25}NO$ (199) dodecanamide

Impossible to identify; only one major fragment (m/z 59) which is the base peak for dodecanamide; may be a long chain amide $RCH_2CH_2CH_2CONH_2$, or a secondary alcohol $CH_3CH_2CH(OH)R$ which would give a base peak of m/z 59 [$CH_3CH_2CH=O^+H$ OR $CH_2=C(OH)N^+H_2$].

#1320 $C_{10}H_{20}O_2$ (172) decanoic acid

Looks like hexadecanoic acid; definitely a higher homolog of #1198.

#1353 $C_{20}H_{42}$ (282) eicosane

Very minor component; unbranched alkane $C_{20}H_{42}$.

#1376 $C_{20}H_{34}O$ (290) 1-naphthalenepropanol, alpha-ethenyldecahydro-alpha,5

Minor component; unidentified.

#1399 No fits found

This is an unidentified but interesting spectrum; it looks like a nitrogen-containing, long chained compound $C_{18}H_{33}N$, containing an alkene or dialkene substituent.

#1428 $C_{18}H_{36}$ (252) 1-octadecene

Identity - virtually certain.

#1487 $C_{18}H_{34}O_2$ (282) (Z)-9-octadecenoic acid

Impossible to identify; related in structure to #1300; probably $CH_3CH_2CH(OH)R$ OR $RCH_2CH_2CH_2CONH_2$ type compound.

#1507 $C_{16}H_{33}NO$ (255) hexadecanamide

Very minor component; identity - high probability to virtually certain.

#1517 $C_{18}H_{36}O_2$ (284) octadecanoic acid

Identity - high probability; this is stearic acid.

#1672 and #1689 $C_{18}H_{35}NO$ (281) (Z)-9-octadecenamide

This is a major component; related in structure to #1507 and #1300; almost certainly an octadecenamide, $C_{15}H_{29}CH_2CH_2CONH_2$

#1719 $C_{22}H_{42}O_4$ (370) hexanedioic acid, dioctyl ester

Identity - high probability.

#1847 $C_{24}H_{38}O_4$ (390) 1,2-benzenedicarboxylic acid, diisooctyl ester

Identity - virtually certain; this is a phthalate; major component.

#1919 $C_{22}H_{46}$ (310) docosane

Peak contaminated by #1847; minor component, unidentified.

#2065 No fits found

Unidentified.

#2092 $C_{30}H_{50}$ (410) 2,6,10,14,18,22-tetracosahexaene, 2,6,10,15,19,23-hexamethyl

Identity - virtually certain; major component; long chain unsaturated hydrocarbon.

#2283 $C_{27}H_{46}O$ (386) (3 beta)-cholest-5-en-3-ol

Steroid; a cholesten-3-ol, $C_{27}H_{46}O$, identity - virtually certain; stereochemistry and position of double bond unknown.

ACETYLATED FRACTION - SEMIQUANTITATIVE DATA

Components Identified With Very High Probability

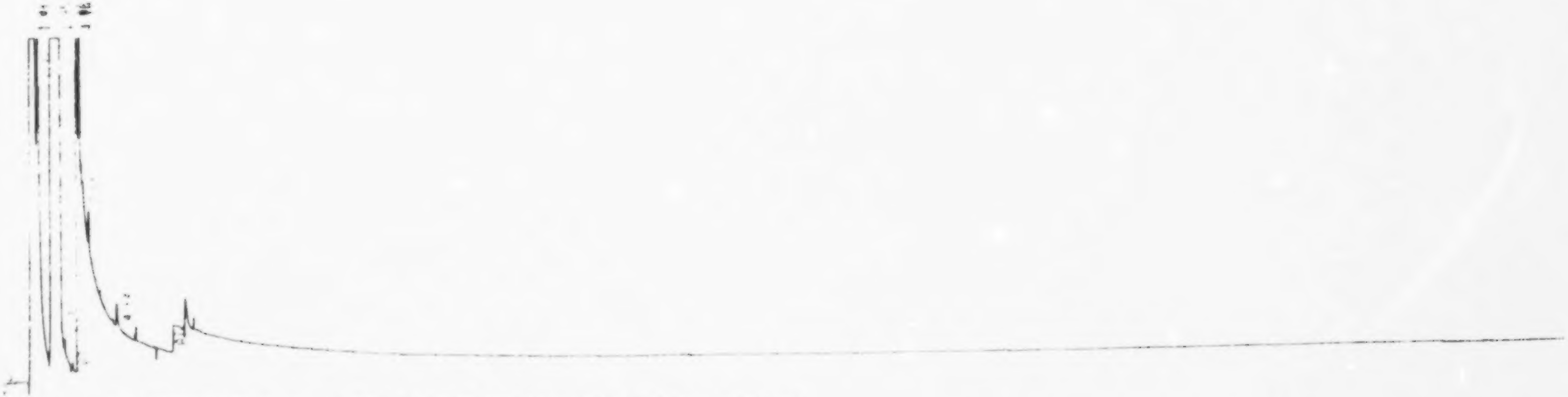
Spectrum #	Component	Approximate Conc. (ug/L)
122	A methylbenzaldehyde (probably 4-methylbenzaldehyde)	1-5
143	Methyl benzoate	1-5
278	alpha-Methylbenzenemethanol acetate	<1
616	$C_{14}H_{30}$ alkane with minor branching (possibly tetradecane)	<1
886	n-Hexadecane	<1
1130	n-Octadecane	<1
1156,1260	Butyl isobutyl phthalate and unidentified phthalate	<1-5
1428	1-Octadecene	5-15
1847	Diisooctyl phthalate	20-30
2092	A $C_{30}H_{50}$ branched polyene	20-30
2283	Cholesten-3-ol (position of double bond unknown)	<1

Components Identified With High Probability

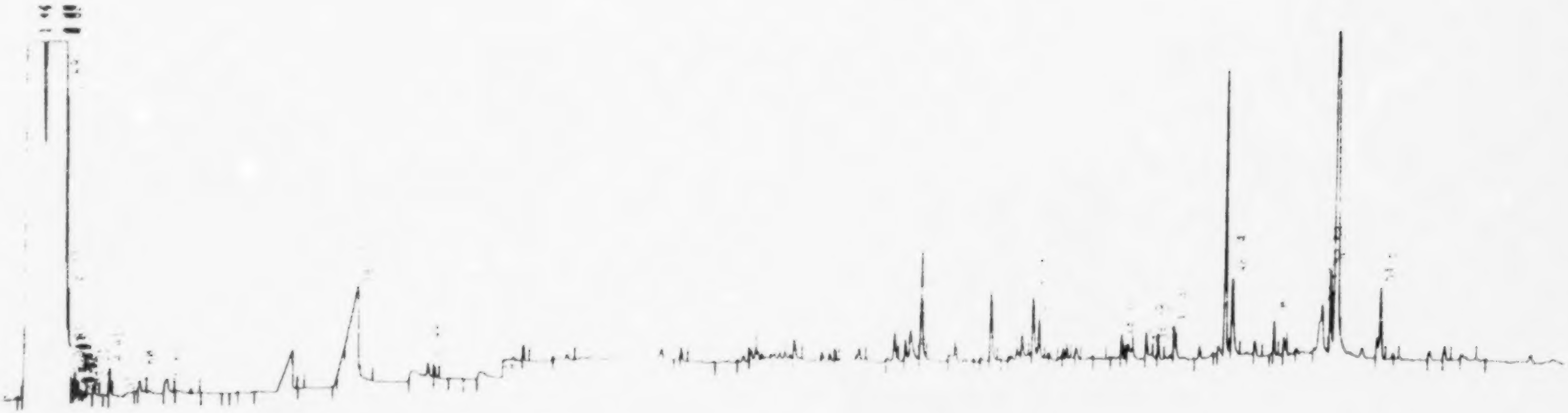
Spectrum #	Component	Approximate Conc. (ug/L)
59	2,4-Pentanedione	<1
101	Acetophenone	1-5
233	Cresol (isomer unknown)	<1
387	A ring methylated benzyl alcohol	5-15
667,671	2,6-bis-(t-Butyl)-2,5-cyclohexadiene-1,4-dione	5-15
811	3,5-Diacetyl-2,6-dimethyl-4H-pyran-4-one	5-15
981,987	2,3-Dihydro-1,1,3-trimethyl-3-phenyl-1H-indene	5-15
1087	Tetradecanoic acid	1-5
1283	9-Hexadecenoic acid	<1
1320	Hexadecanoic acid	5-15
1507	Hexadecanamide	1-5
1517	Octadecanoic acid	1-5
1672,1689	Octadecenamide	20-30
1719	Hexanedioic acid, dioctyl ester	1-5

Components Identified With Lower Probability

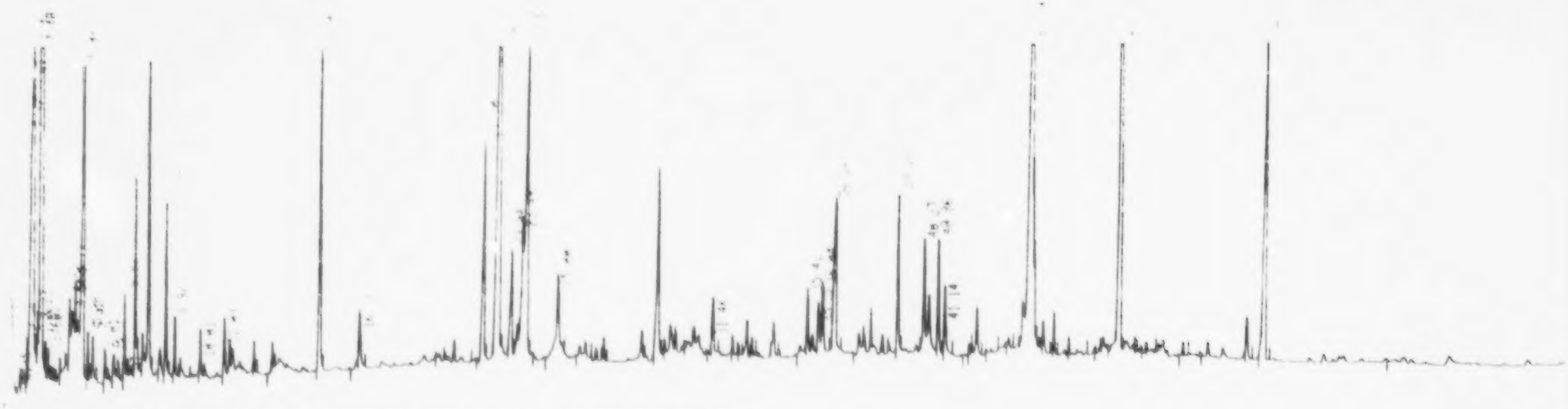
Spectrum #	Component	Approximate Conc. (ug/L)
118	2,4,6-Heptanetrione	1-5
451	Cyclopentylmethyl cyclohexane	1-5
742,752	alpha, alpha-Dimethylbenzyl isopropyl ether	5-15



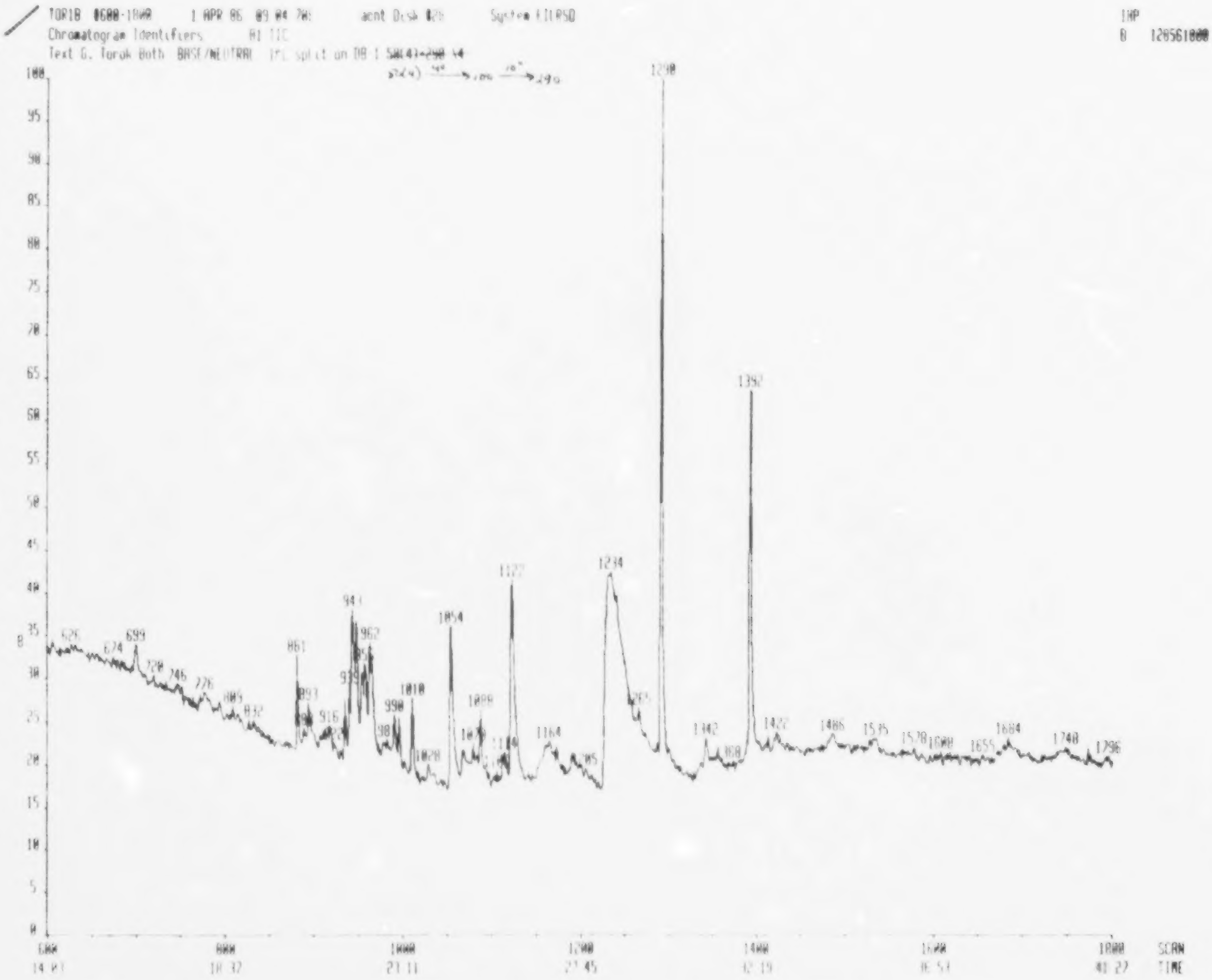
GAS CHROMATOGRAM OF EDMONTON WATER SAMPLE - BASE NEUTRALS FRACTION



GAS CHROMATOGRAM OF EDMONTON WATER SAMPLE - ACID FRACTION



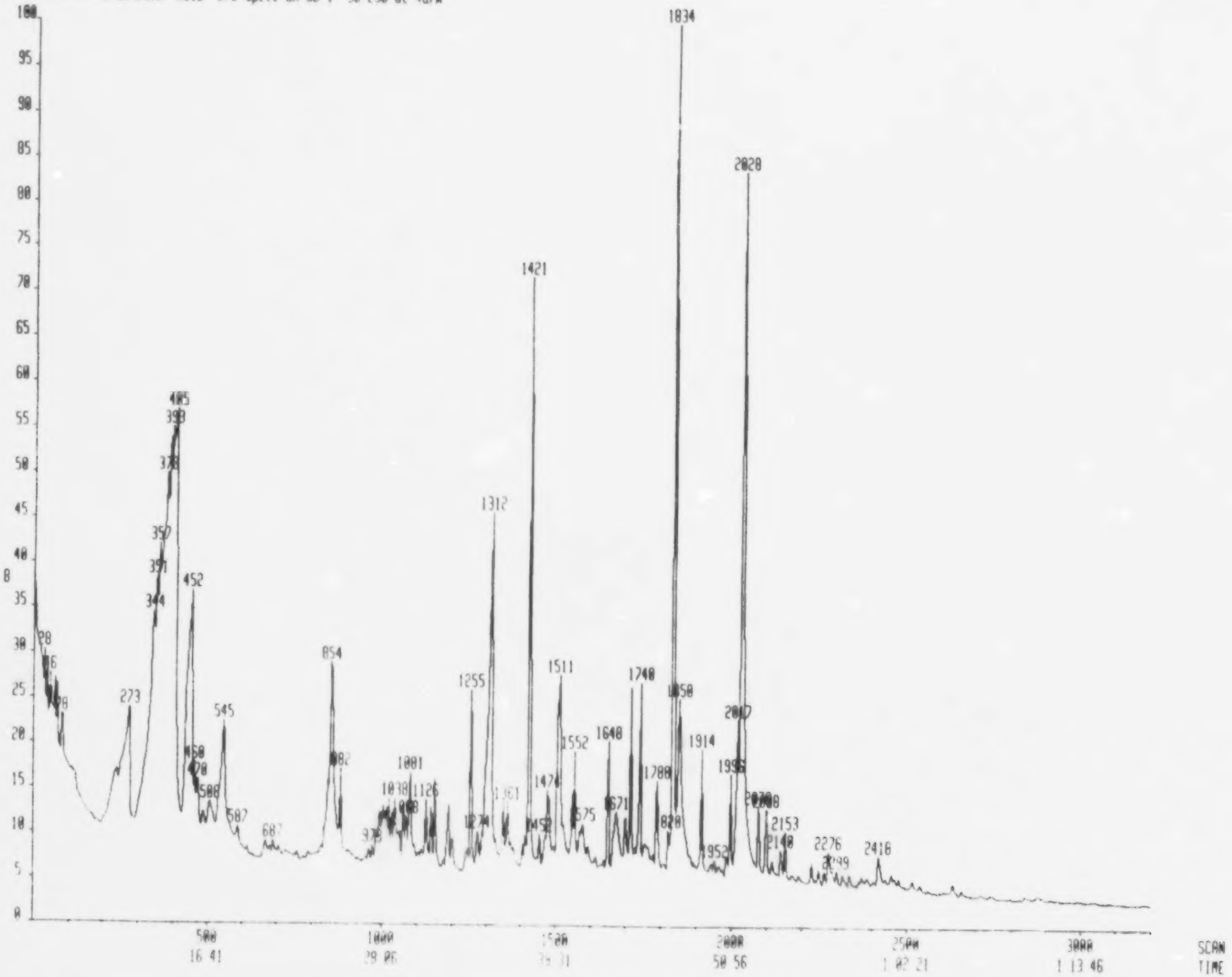
GAS CHROMATOGRAM OF EDMONTON WATER SAMPLE - ACETYLATED FRACTION



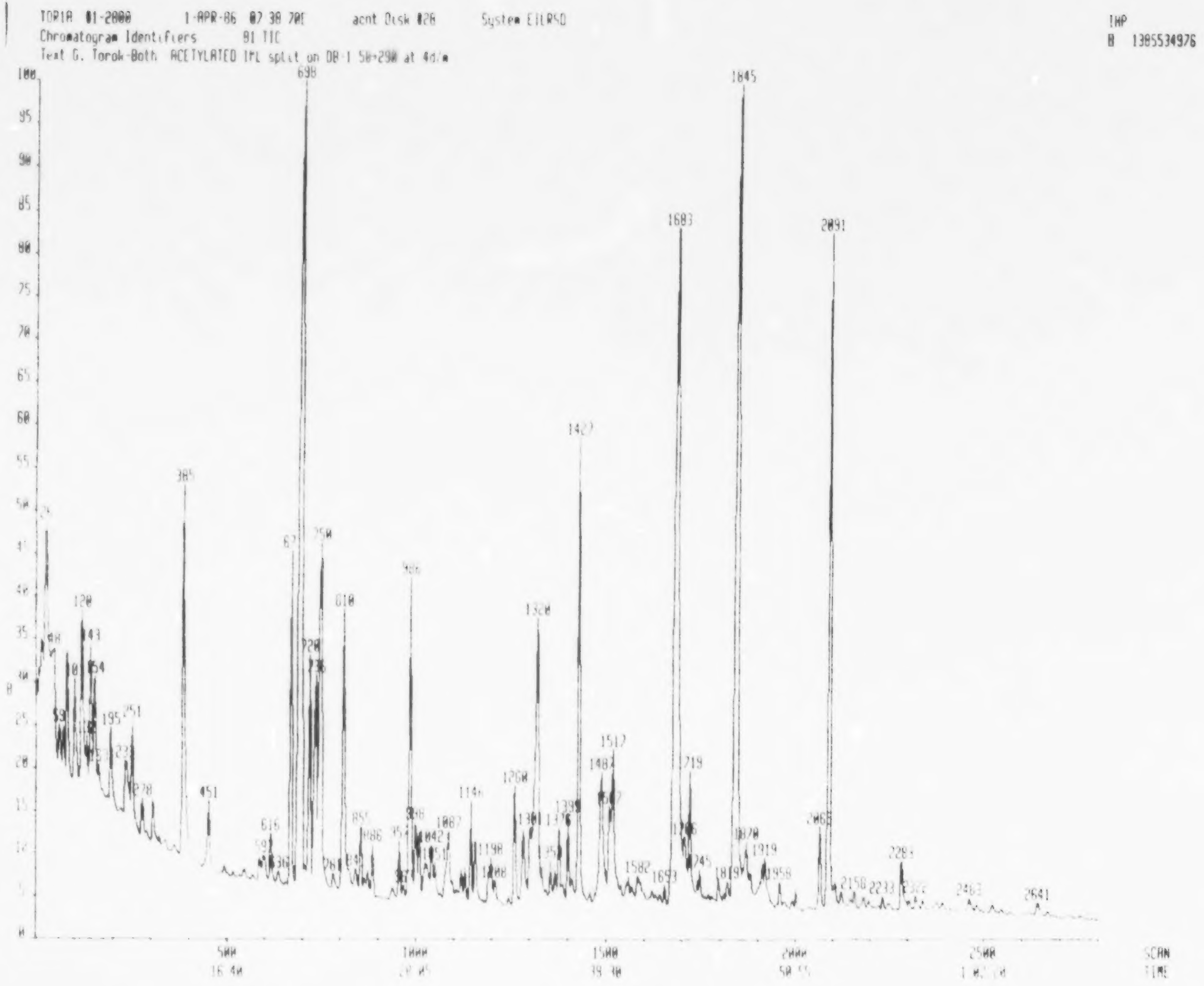
GC/MS TOTAL ION TRACE OF EDMONTON WATER SAMPLE - BASE NEUTRALS FRACTION

TOR31D 01-3200   31-MAR-86 10 29 70E   acnt Disk 020   System EILRSD
Chromatogram Identifiers   01 TIC
Text G. Torok-Roth ACID 2ml split on DB-1 50+290 at 4d/m

IHP
B 942844992



GC/MS TOTAL ION TRACE OF EDMONTON WATER SAMPLE - ACID FRACTION



GC/MS TOTAL ION TRACE OF EDMONTON WATER SAMPLE - ACETYLATED FRACTION

Appendix H

Enviro-Test Laboratories GC/MS Analytical Results for Aldehydes in a Treated Water Sample in Spring 1986

(Sample was in refrigerated storage
for several weeks before analysis)

EtL Enviro-Test Laboratories
A Division of ETL Chemspec Analytical Limited
9936 67th Avenue – Edmonton, Alberta T6E 0P5 Telephone (403) 434-9509

CHEMICAL ANALYSIS REPORT

CITY OF EDMONTON/
WATER AND SANITATION
ROSSDALE WATER TREATMENT PLT.
10155 – 96 AVE
EDMONTON, ALTA

Date: June 10, 1986

ATTN: INNI RASHID

Lab Sample #: 06-86-777 Sampled By: Client

Customer #: P52991-12 Date Received: May '86

Sample Description: 1 water sample B.G. for volatile hydrocarbons by purge and trap.

OVERVIEW:

One sample 18:45, 3/3/86, B.G. and three aldehyde standards were analyzed by purge and trap, GC/MS. The standards gave a relatively poor response on GC/MS however adequate spectra and retention times of each were obtained. The sample was then analyzed for the three target aldehydes.

RESULTS:

COMPOUND	ppb
1) 2-methyl propanal	54
2) 3-methyl butanal	46
3) 2-methyl butanal	134

CERTIFIED BY ____________
Dennis Erickson, Analytical Chemist

Appendix I

GC/MS Priority Pollutants Analysis on Raw and Treated Water from Spring 1986 by Montgomery Labs

(Sample was in transit unrefrigerated after several weeks of refrigerated storage)

MONTGOMERY LABORATORIES
a division of James M. Montgomery, Consulting Engineers, Inc.
555 East Walnut Street, Pasadena, California 91101
(818) 796-9141 / (213) 681-4255 Telex 67-5420

Report of GC/MS Analysis for VOLATILE ORGANICS

Hrudey & Assoc., LTD., Steve E.
14611 Riverbend Rd.
Edmonton, AB T6H 4V5
Attn: Steven Hrudey

Job#/PO#: 437.0656
Workorder#: W07490
Report#: R28702
Phone #: 403-432-5122

Date Sampled: 3/4/86
Date Analyzed: 5/27/86
Date Received: 5/22/86

Lab Number: G59896
Sample I.D.: E. L. SMITH-"RAW WATER"

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS:		
Acrolein	ND	5.0
Acrylonitrile	ND	5.0
Benzene	0.5	0.5
Bromoform	ND	0.5
Carbon Tetrachloride	ND	0.5
Chlorobenzene	ND	0.5
Dibromochloromethane	ND	0.5
Chloroethane	ND	0.5
2-Chloroethylvinylether	ND	0.5
Chloroform	0.5	0.5
Dichlorobromomethane	ND	0.5
1,1-Dichloroethane	ND	0.5
1,2-Dichloroethane	ND	0.5
1,1-Dichloroethene	ND	0.5
1,2-Dichloropropane	ND	0.5
Ethylbenzene	ND	0.5
Methyl Bromide	ND	0.5
Methyl Chloride	ND	0.5
Methylene Chloride	ND	0.5
1,1,2,2-Tetrachloroethane	ND	0.5
Tetrachloroethene	ND	0.5

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

Approved by ____________

APPROVED
QC OFFICER

JUN 2 1986

Report of GC/MS Analysis for VOLATILE ORGANICS

Lab Number:	G59896
Sample I.D.:	E. L. SMITH-"RAW WATER"

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS (continued):		
Toluene	ND	0.5
1,1,1-Trichloroethane	ND	0.5
1,1,2-Trichloroethane	ND	0.5
Trichloroethene	ND	0.5
Vinyl Chloride	ND	0.5
trans-1,3-Dichloropropene	ND	0.5
cis-1,3-Dichloropropene	ND	0.5
trans-1,2-Dichloroethene	ND	0.5
Trichlorofluoromethane	ND	0.5
VOLATILE NON-PRIORITY POLLUTANTS:		
Acetone	4400	0.5
Methylethylketone	ND	0.5
Tetrahydrofuran	ND	0.5
m,p-Xylenes	ND	0.5
o-Xylene	ND	0.5
cis-1,2-Dichloroethene	ND	0.5

Note: Sample arrived in nonstandard bottle containing headspace.

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

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555 East Walnut Street, Pasadena, California 91101
(818) 796-9141 / (213) 681-4255 Telex 67-5420

Report of GC/MS Analysis for VOLATILE ORGANICS

Hrudey & Assoc., LTD., Steve E.
14611 Riverbend Rd.
Edmonton, AB T6H 4V5
Attn: Steven Hrudey

Job#/PO#: 437.0656
Workorder#: W07479
Report#: R28678
Phone #: 403-432-5122

Date Sampled: 3/3/86
Date Analyzed: 5/27/86
Date Received: 5/21/86

Lab Number: G59851
Sample I.D.: E. L. SMITH TREATED (5/3/86)

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS:		
Acrolein	ND	1.0
Acrylonitrile	ND	1.0
Benzene	ND	0.1
Bromoform	ND	0.1
Carbon Tetrachloride	ND	0.1
Chlorobenzene	ND	0.1
Dibromochloromethane	ND	0.1
Chloroethane	ND	0.1
2-Chloroethylvinylether	ND	0.1
Chloroform	6.7	0.1
Dichlorobromomethane	ND	0.1
1,1-Dichloroethane	ND	0.1
1,2-Dichloroethane	ND	0.1
1,1-Dichloroethene	ND	0.1
1,2-Dichloropropane	ND	0.1
Ethylbenzene	ND	0.1
Methyl Bromide	ND	0.1
Methyl Chloride	ND	0.1
Methylene Chloride	ND	0.1
1,1,2,2-Tetrachloroethane	ND	0.1
Tetrachloroethene	ND	0.1

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

Approved by A. Eaton

JUN 2 1986

APPROVED
QC OFFICER

Report of GC/MS Analysis for VOLATILE ORGANICS

Lab Number: G59851
Sample I.D.: E. L. SMITH TREATED (5/3/86)

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS (continued):		
Toluene	ND	0.1
1,1,1-Trichloroethane	ND	0.1
1,1,2-Trichloroethane	ND	0.1
Trichloroethene	ND	0.1
Vinyl Chloride	ND	0.1
trans-1,3-Dichloropropene	ND	0.1
cis-1,3-Dichloropropene	ND	0.1
trans-1,2-Dichloroethene	ND	0.1
Trichlorofluoromethane	ND	0.1
VOLATILE NON-PRIORITY POLLUTANTS:		
Acetone	ND	0.1
Methylethylketone	11	0.1
Tetrahydrofuran	ND	0.1
m,p-Xylenes	0.1	0.1
o-Xylene	ND	0.1
cis-1,2-Dichloroethene	ND	0.1
OTHER COMPOUNDS DETECTED:		
3-Methyl Butanal	(0.3)	
2-Methyl Butanal	(0.2)	

Note: Sample arrived in nonstandard bottle containing headspace.

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

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a division of James M. Montgomery, Consulting Engineers, Inc.
555 East Walnut Street, Pasadena, California 91101
(818) 796-9141 / (213) 681-4255 Telex 67-5420

Report of GC/MS Analysis for
VOLATILE ORGANICS

Hrudey & Assoc., LTD., Steve E.
14611 Riverbend Rd.
Edmonton, AB T6H 4V5
Attn: Steven Hrudey

Job#/PO#: 437.0656
Workorder#: W07479
Report#: R28677
Phone #: 403-432-5122

Date Sampled: 3/3/86
Date Analyzed: 5/27/86
Date Received: 5/21/86

Lab Number: G59850
Sample I.D.: E. L. SMITH (3/3/86)

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS:		
Acrolein	ND	1.0
Acrylonitrile	ND	1.0
Benzene	0.1	0.1
Bromoform	ND	0.1
Carbon Tetrachloride	ND	0.1
Chlorobenzene	0.2	0.1
Dibromochloromethane	0.2	0.1
Chloroethane	ND	0.1
2-Chloroethylvinylether	ND	0.1
Chloroform	5.6	0.1
Dichlorobromomethane	0.2	0.1
1,1-Dichloroethane	ND	0.1
1,2-Dichloroethane	ND	0.1
1,1-Dichloroethene	ND	0.1
1,2-Dichloropropane	0.1	0.1
Ethylbenzene	ND	0.1
Methyl Bromide	ND	0.1
Methyl Chloride	ND	0.1
Methylene Chloride	ND	0.1
1,1,2,2-Tetrachloroethane	0.4	0.1
Tetrachloroethene	0.1	0.1

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

Approved by A. Eaton

JUN 2 1986

APPROVED
QC OFFICER

Report of GC/MS Analysis for VOLATILE ORGANICS

Lab Number: G59850
Sample I.D.: E. L. SMITH (3/3/86)

Compound	Concentration (micrograms/liter)	Detection Limit (micrograms/liter)
VOLATILE PRIORITY POLLUTANTS (continued):		
Toluene	0.2	0.1
1,1,1-Trichloroethane	ND	0.1
1,1,2-Trichloroethane	0.1	0.1
Trichloroethene	0.1	0.1
Vinyl Chloride	ND	0.1
trans-1,3-Dichloropropene	0.1	0.1
cis-1,3-Dichloropropene	0.2	0.1
trans-1,2-Dichloroethene	ND	0.1
Trichlorofluoromethane	ND	0.1
VOLATILE NON-PRIORITY POLLUTANTS:		
Acetone	4.3	0.1
Methylethylketone	49	0.1
Tetrahydrofuran	ND	0.1
m,p-Xylenes	ND	0.1
o-Xylene	ND	0.1
cis-1,2-Dichloroethene	ND	0.1
OTHER COMPOUNDS DETECTED:		
3-Methyl Butanal	(1.0)	
2-Methyl Butanal	(0.6)	

Note: Sample arrived in nonstandard bottle containing headspace.

ND: Not Detected
NA: Not Analyzed
NQ: Not Quantified; detected below minimum detection limit
(): Tentative value only

Preliminary Draft for Comment

CONTINUOUS WATER QUALITY MONITORS FOR WATER INTAKE PROTECTION FROM SPILLS

a task report for

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

by

Steve E. Hrudey & Associates Ltd.

July 1986

Preliminary Draft

1. INTRODUCTION 1

2. PHYSICAL / CHEMICAL MONITORS 2

3. BIOLOGICAL MONITORS 3

4. RECOMMENDATIONS 11

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1. INTRODUCTION

Water intakes located on surface water sources are inherently vulnerable to chemical spill contamination. The degree of concern directed to this vulnerability should depend upon the specific circumstances which exist. In the case of Edmonton, a review of spill potential (Hrudey-1, this Volume) indicates that the E.L.Smith intake is not exposed to major risk because of the limited upstream development. The existing Rossdale intake is very vulnerable because of its location downstream and impact from 85 storm sewer outfalls.

Given the situation at Rossdale, the need for spill protection is readily apparent. The question which arises is could the provision of continuous monitors at this intake provide enough reliable warning to make relocation of the intake unnecessary. To deal with that question, a literature review was performed and equipment suppliers were contacted to determine what equipment and experience may be applicable to Edmonton. In addition, considerable documentation was available on biological monitors because this option has been actively considered by Edmonton.

Although substantial information was reviewed, relatively little information was directly relevant to Edmonton's particular circumstance. Accordingly, the following discussion will only highlight a few publications relevant to the question posed above. This report will be considered a preliminary draft and as further information is acquired, the analysis will be updated for the final report.

2. PHYSICAL / CHEMICAL MONITORS

Hunt and Wilson (1986) and Briggs (1979) provide an excellent discussion of automated analyzers and specific applications for continuous monitoring of raw water. They noted the great interest which exists in these applications but could cite only one application at an operational water works (Sunderland and South Shields Water Company, Sunderland, U.K.). Most continuous monitors used on rivers are measuring inorganic parameters, such as ammonia, dissolved oxygen, pH, and conductivity. For organic analyses, which would represent the major concern for Edmonton, they could mention only UV absorbance monitoring at 250-254 nm. They report that continous UV absorbance monitoring is feasible with the proviso that compensation for interference by turbidity may be necessary. Presumably, concurrent turbidity analysis would be necessary.

The main limitation of the UV approach is that it will not be sensitive enough for low levels of many organics which the treatment plant would want to be warned about. However, experience at Edmonton has shown that UV absorbance can provide an excellent indication of high dissolved organic carbon as occurs with rural spring runoff.

Wallwork and Ellison (1983) state, in general terms what an automated system should be capable of, namely:

-provide early warning of the presence of individual, anticipated pollutants and ideally for any pollutant;

-take a discrete sample of the raw water in response to a water quality alarm and for subsequent laboratory examination;

-transmit data to a point or points in the revelant control system for subsequent display and, if necessary, remedial action; and

-operate unattended, other than for occasional attention, in what may be a non-laboratory environment.

These authors have been operating a continuous river monitoring station since 1978.They measure: ammonia; dissolved oxygen; pH; temperature; conductivity; river level; and fish activity. They monitor the ammonia mainly to determine chlorine demand and the pH, dissolved oxygen and temperature are mainly relevant to the health of their fish. Conductivity has been found useful for estimating alkalinity, which is an interesting application for process control purposes.They originally attempted monitoring for dissolved organic carbon but were unsuccessful. The instrument is now being used successfully for monitoring treated water at a pilot plant so the difficulties presumably arose from the raw water.

The impression provided by Wallwork and Ellison is that only their fish monitor is really relevant for organic chemical spill protection.

Davenport et al.(1981) describe an electrochemical organic content (EOC) analyzer intended for continous organics monitoring.The technique measures the capacitance of an electrode - solution interface when an organic is adsorbed to an indicating electrode surface. Since organics adsorb to a different degree, the response of the EOC analyzer will vary according to the particular organic in the sample being tested.Consequently, the analyzer will be specific (ie. only able to detect the presence of one organic at a time) depending on the electrical potential chosen. By properly choosing the electrical potential (between the indicating and reference electrode) one can optimize the sensitivity of the indicating electrode to adsorption of the organic of interest. The optimum potential probably has to be determined under laboratory conditions for each organic of interest (ie. organic of interest diluted in lab grade water, free of interferences).

The EOC analyzer gives a readout (dimensionless) that has to be manually calibrated using lab prepared standards. The EOC is still very much in the research/experimental development stage.Interferences such as organic solids, inorganics co-adsorption from other organics and the applicability to natural humics were not discussed.

Although this device may eventually have some role in continous monitors for protection from organic spills, it is not too relevant to Edmonton's current needs.

Another possibility which was not encountered in the water supply literature, but which may be applicable to Edmonton is fluorometry. Because many of Edmonton's spill concerns relate to hydrocarbons and because polynuclear aromatic hydrocarbons fluoresce, the potential for a useful detector exists. Edmonton currently monitors volatile organics by GC on a regular basis so the measurement of non-volatile, polynuclear hydrocarbons would be complementary.

Continous fluorometers are well developed for rugged field applications in dye-tracing studies. The instrumentation is also not very expensive compared to devices like total organic carbon analyzers.

Finally, automated organic carbon analysis may offer some promise. In particular, the purgeable organic carbon analysis may provide a more rapic surrogate for the volatile organics currently measured by purge and trap GC. However, because the instrument is so expensive, extensive trials would be necessary before any commitments were made.

3. Biological Monitoring Systems

The probability of detecting unusually high concentrations of petroleum related hydrocarbons, or pesticide compounds (resulting from a spill), prior to their entry into the water treatment system is presently low.

What may be required, is a so-called "biomonitoring system." Specifically, a system can involve two types of biological assay, these being, bioaccumulation and fish physiography.

I.E.C. (1981) describes the generalized fish physiograph system in the following manner:

> "free-swimming fish are confined individually in monitoring cells. The microvolt bioelectric potentials emitted as they respire are received by two submerged hard wire electrodes affixed to the ends of the holding cells and are carried along shielded conductors to high gain frequency selective amplifiers and frequency filters for processing. At designated intervals, a mechanical switching device or programmed multiplexer pass the amplified signals to either a hard copy recording system for manual interpretation of the complete respiratory waves or to an A/D converter interfaced with a computer to compile and evaluate the respiratory data. If the fish activity deviates beyond certain critical limits, alarms are activated and remedial action may be taken to restore water quality.

The use of fish, in detecting contaminants in water supplies is not a recent phenomenon. Simple biomonitoring systems based on visual examination of fish in a flow-through tank were initially used (Henderson, and Pickering, 1963). Technological advancements and the need for automation (24 hour surveillence) resulted in a diversification of the monitoring technique. Physiographic systems have utilized fish responses in terms of Locomotor activity (Cripe, et. al., 1975, Morgan, 1979, and Waller and Cairns, 1972) based on either current frequencies produced by fish movement or by photocells using either visible or infra-red light. In addition, ultrasonic sound has been used to record locomotor activity (Scherer, and van der Veen, 1982).

The ability of fish to maintain themselves in a current has also been used (MacLeod and Smith, 1966), as well as changes in heart rate (Venables and Smith, 1972), or oxygen consumption (Davis, 1973, O'Hara, 1971, and Waiwood, and Johansen, 1974).

Venillation rate or coughing (purging of the gills) (Morgan, 1977, and I.E.C., 1981.) has been used quite successfully in monitoring for a variety of compounds. In this system electrodes are placed at either end of the exposure chamber, these electrodes are sensitive to cyclic changes in micro-volt electric potential which are caused by the breathing (opercular) movements of the fish (Spoor, et. al. 1971, Heath, 1972). Refinements have since concentrated on amplifying and filtering the signal, together with computer assisted continuous monitoring systems (Gruber, et. al. 1977, and B.M.I. 1982).

The use of fish respiratory monitors appears to be among the most sensitive of the above indicators. A predefined range of opercular rhythms is incorporated into the computer program, any deviation from this interval (generally in excess) sets off an alarm system to alert the operator(s) . Depending on the sophistication of the system, the software can be set up to; produce printouts of the incoming alarm data, activate any automatic water sampling devices, and initiate phone calls to required personnel, in order to alert them to the alarm condition (Gruber, and Walker, 1984).

It would appear that these systems might be useful as an early warning system for the pickup of chemical slugs (resulting from spills) at the sub-lethal to toxic level.

Although the reliability of the system appears to be improving with new technology, environmental and innate behavioral responses of the fish can result in false alarms (for example, di-urnal changes in behavior patterns; I.E.C., 1981). Individual variability in ventilatory behavior is also a major problem in terms of establishing the alarm limits of the system. For this reason, fish almost have to be monitored on an individual basis (one-to-a-tank). The monitoring system as a whole would therefore require several exposure tanks for statistical validity. Data from I.E.C. (1981) indicate that the ventilatory response between individuals and secondarily between compounds can vary widely. This in turn creates the need for control fish in order to help suppress an alert response which may be due to factors unrelated to water quality (which in turn raises the question of what water do you put the control fish in when you are examining a drinking water supply).

Proper compilation of this incoming data makes computer assistance manditory, in order that a statistically valid "decision" can be calcualted which would result in a true alarm situation (if, in fact, the variability problem can be overcome). Obviously, the more sophisticated the system the higher the capital costs involved (presently it would appear that equipment requirements alone for opercular monitoring system described above would be in the neighborhood of $35,000 to $40,000 CAN., 1983 dollars). The capital costs of such a system, which would not be modest, would easily be matched by the personnel + yearly maintenance costs involved. Therefore, false alarms on a regular basis would not be desireable due to the personnel cost alone.

Although these systems have found applications for the monitoring of industrial effluents (Morgan, et. al., 1983, Fisher, 1982, B.C. Research, 1978), there has been only the report by Wallwork and Ellison (1983) for successful biomonitory use by a drinking water utility. It would appear that the system would be most effective for a treatment plant which experiences poor raw water quality on a regular basis. In their summation of seven different fish physiograph systems, I.E.C. (1981) indicated that,

> "organic chemical concentrations required to elicit a respiratory resonse by fish were several orders of magnitude higher than organic concentrations found in the drinking water supply at Brantford. Fish did not alter their respiratory activity to change in levels of organics at Brantford.

> However, fish respiration could be used to monitor organic levels if ambient concentrations are significantly higher than levels in waters presently measured in the Grand River."

It appears that the so-called biomonitoring systems are still at the point of being refined so as to be useable in a drinking water situation. Although they are certainly a means to establishing continuous water quality monitoring for treated industrial effluents, their practicality for a water source is still uncertain. Calgary is currently evaluating a fish monitoring device and their progress should be followed with interest.

Of related interest is the fact that the University of Alberta Dept. of Zoology uses the Rossdale treated water as is, except for pH adjustment and addition of thiosulphate to remove chlorine, to hold all of its experimental fish.

4. RECOMMENDATIONS

1. The utility of UV absorbance monitoring for detecting dissolved organic carbon associated with waterborne humic materials during spring rural runoff has been sufficiently proven by Edmonton Water Lab staff that installation of a continuous UV monitor at an upstream location, like Devon, should be attempted. The need for concurrent turbidity monitoring will need to be assessed.

2. The low cost and high potential for continuous fluorescence monitoring warrants a trial at Rossdale to determine if the method is sufficiently sensitive to respond to hydrocarbons at the levels encountered at Rossdale.

3. Edmonton should seek to evaluate the potential for using purgeable organic carbon as a more rapid surrogate for their volatile organics method by GC. A cooperative program with the Department of Civil Engineering at the University of Alberta would be cost-effective as they have recently acquired this expensive instrumentation.

4. Edmonton should watch carefully the progress at Calgary using a fish monitor to determine from local experience whether these devices can justify the expense involved by providing reliable intake protection.

5. Edmonton should not rely on any of these continuous monitoring alternatives rather than seeking to improve the quality of raw water at Rossdale by relocating the intake.

REFERENCES:

B.C. Research. 1978. Avoidance reactions of salmonids to pulpmill effluents. Prepared by B.C. Research, Vancouver, for E.P.S., Environment Canada, Ottawa. CPAR Project Report **688-1**.

Briggs, R. 1979. The continuous monitoring of raw water intakes. J. of the Inst. of Water Eng. and Scientists. 33(1): 84 - 94.

B.M.I. 1982. A mobile biological early warning system for the automatic and continuous assessment of water and wastewater quality. Preparted by Biological Monitoring, Inc., Christiansburg, Virginia.

Cripe, C.R., J.H. Cripe, and J.R. Livingston. 1975. Apparatus for quantitative determination of locomotor activity patterns of aquatic organisms using infrared light-emitting diodes. *J. Fish. Res. Board. Can.* **32**:1884-1886.

Davis, J.C. 1973. Sublethal effects of bleached kraft pulp mill effluent on respiration and circulation in sockeye salmon (*Oncorhynchus nerka*). *J. Fish. Res. Board Can.* **30**:369-

Davenport, R.J., R.A. Wynveen, and W.J. Cooper. 1981. Continuous, automated, and low - cost monitoring of the organic content in water. Journal AWWA. 73(9): 555 - 558.

Fisher, J.N. 1982. Employing acute and subacute toxicity measurements in on-site biomonitoring studies. From the Proceedings of the Technical Association of the Pulp and Paper Industry, March/82.

Gruber, D., J. Cairns, Jr., K.L. Dickson, R. Hummell, III, A. Maciorowski, and W.J. van der Schalie. 1977. An inexpensive noise-immune amplifier designed for computer monitoring of ventilatory movements of fish and other biological events. *Trans. Am. Fish Soc.* **106**:497-

Heath, A.G. 1972. A critical comparison of methods for measuring fish respiratory movements. *Water Res.* **6**:1-7

Henderson, C., and Q.H. Pickering. 1963. Use of fish in the detection of contaminants in water supplies. *J. A.W.W.A.* **51**:715-720.

I.E.C. 1981. Biomonitoring of Organic Compounds in drinking water. Prepared for the Ontario Ministry of the Environment by International Environmental Consultants Ltd., Toronto, Ontario. O.M.E. Project. **2076.1**:230pp.

MacLeod, J.C. and L.L. Smith, Jr. 1966. Effect of pulpwood fiber on oxygen consumption and swimming endurance of the fathead minnow, *Pimephales promelus* . *Trans. Am. Fish. Soc.* **95**:71-

Morgan, W.S.G. 1977. An electrode system to monitor the effects of changes in water quality on fish opercular rhythms. (In) Biological Monitoring of Water and Effluent Quality. Edited by J. Cairns, K.L. Dickson, and G.F. Westlake. *A.S.T.M. STP* **607**:38-55.

Morgan, W.S.G. 1979. Fish locomotor behavior patterns as a monitoring tool. *J. Wat. Pollut. Control Fed.* **51**:580-589.

O'Hara, J. 1971. Alterations in oxygen consumption by bluegills exposed to sublethal treatment with copper. *Water Res.* **5**:321-.

Scherer, E. and B. van der Veen. 1982. An automated beam actograph for laboratory and field use. Can. Tech. Rep. Fish. Aquat. Sci. 1137:15pp.

Spoor, W.A., T.W. Neiheisel, and R.A. Drummond. 1971. An electrode chamber for recording respiratory and other movements of free-swimming animals. *Trans. Am. Fish. Soc.* **100**:22-.

Waiwood, K.G. and P.H. Johansen. 1974. Oxygen consumption of the white sucker (*Catostomus commersoni*) in lethal and nonlethal levels of the organochlorine insecticide, methoxychlor. *Water Res.* **8**:401-

Wallwork, J.F. and G. Ellison. 1983. Automated sensing systems for water resourse pollution warning and treatment process control. Aqua. 6: 313 - 320.

Waller, W.T. and J. Cairns, Jr. 1972. The use of fish movement pattern to monitor zinc in water. *Water Res.* **6**:257-269.

Evaluation of Advanced European Drinking Water Treatment In Relation to Practice at Edmonton

A report prepared for

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1. INTRODUCTION

In the fall of 1985 the City of Edmonton and Alberta Environment retained S.E. Hrudey and Associates, Ltd. to conduct an independent assessment of the quality of Edmonton's drinking water.

Included in this study was to be a comparison of Edmonton's raw and treated water quality to that for selected locations in Europe, and an assessment of advanced European drinking water treatment practice. S.E. Hrudey and Associates retained the writer to complete this task.

2. SCOPE OF WORK

The terms of reference for this component of the overall study are given in Appendix 1. Briefly, they involved visiting the E.L. Smith and Rossdale treatment plants and critiquing their operation; obtaining raw and treated water quality data for selected European locations; and reviewing advanced water treatment practice at three or more European locations and recommending processes for possible study in Edmonton.

This report provides a detailed review of three advanced European water treatment plants. Raw and finished water quality data for the three locations are provided in Appendix 3.

Site visit reports for the Edmonton plants are given in Appendix 4. In addition, Dr. Snoeyink's site visit report was reviewed and comments forwarded to him. The writer's particular process concerns following the site visits were:

1. the simultaneous addition of a number of process chemicals,
2. the high turbidity of water going onto the filters at Rossdale, and
3. the need for overall process optimization, particularly with respect to

organics removals.

These matters are addressed in the recommendations at the end of this report.

3. BASIS OF EUROPEAN PLANT SELECTION

The following principles served as guides for selection of the European plants to be evaluated:

1. The plants chosen should be internationally recognized as representing advanced European practice for surface water treatment.
2. Each plant should be in a different country to reflect differences in national practices. The countries included should be West Germany, France, and either Switzerland or the Netherlands.
3. The plants should be generally comparable to Edmonton in size and in raw water quality.
4. To the extent possible the plants should be ones which the writer had visited.
5. The locations should have had some experience with taste and odour problems.
6. The locations chosen should have information generally available at the required level of detail.

With these principles in mind the following plants were chosen for study: Langenau in West Germany, Zurich in Switzerland and Méry-sur-Oise in the Paris suburbs in France. The Netherlands was eliminated because advanced plants there with which the writer was familiar are all near the mouth of the Rhine or Meuse, both of which are far more heavily contaminated than the North Saskatchewan. For similar reasons the German plant chosen was not on the Rhine. A brief introduction to each plant follows:

1. West Germany

The plant at Langenau provides part of the supply for the city of Stuttgart. It is located on the upper reaches of the Danube which at that point is influenced primarily by agricultural and municipal wastes with little industry. The plant utilizes a novel compact flocculation step, ozone, and granular actived carbon and produces an excellent quality water. When the writer was there in 1983 they were also piloting a softening process for possible future use.

2. Switzerland

The Zurich plants obtain water from Lake Zurich. A taste and odour episode with phenols approximately fifteen years ago led to the abandoning of chlorine in favour of ozone. The Zurich plants currently use chlorine, chlorine dioxide and GAC in addition to ozone. Zurich is the only location which the writer has not visited.

3. France

The Méry-sur-Oise plant is one of several serving the Paris suburbs. Along with the others, it uses ozone and granular activated carbon to provide multi-step treatment including biological activity on the carbon. All of the Paris suburban plants experience generally worse raw water quality than Edmonton, but given the site selection criteria and study constraints it was not possible to find a French plant with raw water quality comparable to Edmonton's.

A sample of the request for information sent to the three plants may be found in Appendix 2.

4. DETAILED PLANT DESCRIPTIONS

4.1 Zurich, Switzerland

Except where indicated the information in this section is taken from Schalekamp (1986).

4.1.1. Source of Supply

Zurich and its environs receive approximately 70% of their water from Lake Zurich. This amount is supplied by the Lengg and Moos plants. The remainder of the supply comes from treated groundwater (20%) (Hardhof) and spring water (10%). The groundwater and spring water supplies are not considered in this review.

Data for selected raw water quality parameters are provided in Table 1. Detailed data are given in Appendix 3.

Table 1 shows a raw water of generally excellent quality, with little variation in all major parameters. Turbidity is low, pH is in the alkaline range, and the water is slightly hard. Ammonia, phosphate-phosphorus and organic carbon are all low. The water is also of excellent bacterial quality.

4.1.2 Size of System

In the 1983 report of the Wasserversorgung Zürich (Zurich water supply) the population of the city is given as 366,000. Another 318,000 persons in areas to which water is supplied under contract ("contract partners") brings the total population served to 684,000. The volumes of water provided to the city and contract partners respectively in 1983 were 62.19 and 11.58 million m^3.

The daily per capita consumption calculated for the city is 466 litres or approximately 104 Imperial gallons. The amount for the contract partners is considerably less, 100 litres per capita per day or approximately 22 Imperial gallons. This figure presumably represents only domestic consumption and may

Table 1. Selected Raw Water Quality Parameters for Zurich, Switzerland (1985 data)

Parameter	Units	Lengg Plant			Moos Plant		
		Avg.	Min.	Max.	Avg.	Min.	Max.
Temperature	°C	5.8	4.3	7.1	5.8	3.0	9.0
Turbidity[a]	FTU	0.28	0.17	0.50	0.37	0.16	0.72
pH		7.85	7.62	7.99	7.86	7.76	8.05
Conductivity	µS/cm	260	245	280	260	255	270
Total hardness	°fr	13.7	13.2	14.1	13.7	13.2	14.0
Total hardness	mg/L $CaCO_3$[b]	137	132	141	137	132	140
Chloride[c]	mg/L	4.3	4.1	4.4	5.7	4.7	8.0
Dissolved oxygen	mg/L	8.9	6.5	10.6	8.5	6.9	10.1
NH_3-N[a]	µg/L	<5	<5	<5	<5	<5	5
$PO_4^{-}P$[a]	µg/L	53	47	64	52	41	59
DOC	mg/L	1.20	1.05	1.30	1.28	1.05	2.15
Total PAH	ng/L	24.0	8.1	46.8	21.0	-	-
Total Coliforms	per 100 mL	4	0	40	8	0	30

[a] After chlorine and chloride dioxide addition (Moos plant); after chlorine addition (Lengg plant).

[b] Assuming that °fr stands for French degree of hardness
1 French degree of hardness = 10 mg/L $CaCO_3$

[c] After chlorine and chlorine dioxide addition in Moos plant only.

include areas which are only partly serviced.

4.1.3 Process Description

For each plant a brief description of the historical development is given to provide a background for the treatment presently used.

1. Lengg Plant

The Lengg Plant is located on the right or east bank of the lake approximately 60 m above lake level. Up to 1960 treatment consisted of slow filtration. In that year the capacity was increased to 80,000 m^3 per day. All water was given rapid filtration and then the flow was split. Up to 40,000 m^3 per day could be passed through the slow sand filters; the remainder was chlorinated and the two streams were then blended.

During this time increasing bacterial contamination was noted in the raw water. On September 20, 1967 there was a large phenol spill in the lake. Water quality to half the population, presumably those areas served by the Lengg plant, was seriously impaired. Following this chlorine was replaced with ozone for disinfection. This represented the first use of ozone in Zurich.

The use of ozone produced significant bacterial regrowth. After 23 days in operation bacterial counts of up to 100,000 per mL were obtained in the distribution system. This was attributed to the formation of biodegradable organic compounds by the ozonation. Following this the ozonation facility had to be closed down for another reason, namely because of the lack of an ozone destruction capability. Such capability has since been provided.

In 1971 this plant underwent a threefold capacity expansion. Quality improvements were also required because of deteriorating lake water quality caused by a variety of factors. Another problem was that the migratory shell DPP (Dreissena Polymorphy Pallas) appeared in large numbers in the lake between 1970 and 1975. The larvae of these shells present a problem, remaining suspended in the water for two weeks before settling. Chlorine was required to destroy the larvae. Studies showed that with prechlorination to a residual of 0.5 mg/L the larvae were destroyed within 13 days in rapid filtration. It is not clear how the 13 day contact time would be obtained unless the larvae were retained on the filter and were in contact with the chlorine for that time. For dechlorination a layer of GAC was placed on the slow sand filters.

Process development work was performed between 1971 and 1975. The new process used as of 1975 (Figure 1) consisted of the following steps:

prechlorination,
microfloccuation with aluminum sulphate,
pH adjustment with lime,
dual media rapid filtration,
ozonation,
granular activated carbon,
slow filtration, and
chlorine dioxide for distribution system residual.

A problem which became apparent at this time was trihalomethane formation. Up to 25 µg/L were present following prechlorination, with chloroform accounting for 80%. These levels were reduced by ozonation and GAC.

2. Moos Plant

The Moos plant initially began operation in 1914 with primary and slow filters. Its present capacity is 120,000 m^3/day of lake water and up to 30,000 m^3/day of spring water. The process is basically the same as at Lengg except that the lake water is mixed with spring water prior to ozonation (Figure 2). Another difference is that chlorine and chlorine dioxide are used

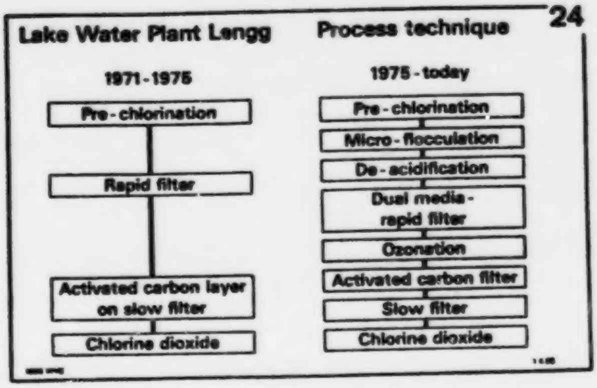


Figure 1 Process Flowsheet for Lengg Plant, Zurich

Source: Schalekamp (1986)

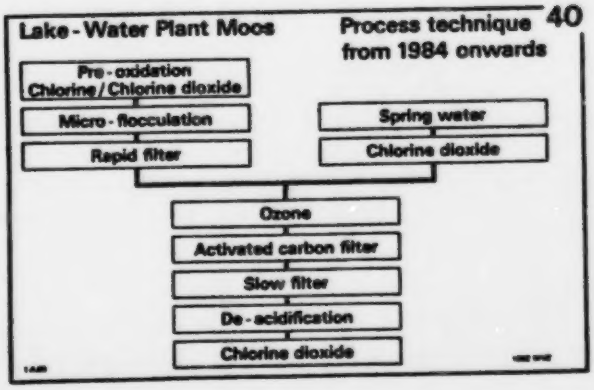


Figure 2 Process Flowsheet for Moos Plant, Zurich

Source: Schalekamp (1986)

together for preoxidation and the pH adjustment is at the end.

The mixture of oxidants is a process which was developed at the Hardhof ground water plant. It was found that by using 0.3 mg/L of chlorine dioxide and 0.5 mg/L of chlorine as a predisfectant both trihalomethane and chlorite concentrations could be minimized. To-date results at Hardhof have shown less than 1 µg/L trihalomethanes and less than 0.1 mg/L of chlorite and chlorate after ground passage. This mixture has also been found to be better than chlorine alone for destruction of the larvae.

Problems were encountered with degassing of chlorine dioxide in the closed primary and rapid filters. This resulted in significant corrosion problems.

In 1982 the old preliminary filters with a daily capacity of 60,000 m^3 were reconstructed to multi-media rapid filters. Because of problems with the chlorine dioxide degassing it was impossible to meet the required standard for chlorine dioxide in air of 0.1 ppm (Maximum Working Space Concentration). The filters therefore were partitioned off and presumably ventilation was provided.

4.1.4 Process Performance

Table 2 summarizes the effect of treatment on various water quality parameters for both the Lengg and Moos plants in 1985. It is evident that extremely low finished water turbidities are achieved. With respect to overall organic parameters, the UV-extinction value is reduced by at least a factor of three, while dissolved organic carbon is reduced by less than 50%. Little reduction is achieved in THMs, but they are at a low level initially.

Overall, the finished water is of excellent quality.

Table 2. Effect of Treatment or Selected Water Quality Parameters at Zurich, Switzerland (Average values, 1985).

		Lengg Plant			Moos Plant		
Parameter	Units	Raw Water	Rapid Sand Filtrate	Finished[a] Water	Raw Water	Rapid Sand Filtrate	Finished[a,b] Water
Turbidity	FTU	0.28	0.06	0.04	0.37	0.10	0.04
NH_3-N	μg/L	<5	-	<5	<5	-	<5
Phosphate-P	μg/L	53	-	44	52	-	21
Iron	μg/L	20	5	5	30	15	5
UV-extinction	254 nm/1m	3.0	2.3	1.0	2.9	2.2	0.8
DOC	mg/L	1.20	1.20	0.85	1.28	1.08	0.65
Total THMs	μg/L	5.1[c]	-	5.2	3.1[d]	-	1.5
Total PAH	ng/L	24.0	-	21.5	21.0	-	21.7
Total Coliforms	per 100 mL	4	0	0	8	0	0

[a] With chlorine dioxide added.

[b] A mixture of 72% lakewater and 28% spring water.

[c] After chlorine addition.

[d] After chlorine plus chlorine dioxide addition.

4.1.5 Regulations on Treated Water Quality

Regulations for selected parameters are summarized in Table 3, in the column headed SLB. The current regulation is 25 µg/L for trihalomethanes, while the proposed regulation is 10 µg/L for trihalomethanes plus organic solvents. The current regulation is a maximum concentration of 0.3 mg/L for chlorite plus chlorate in treated water, while the proposed stipulation is a maximum dose of 0.2 mg/L for chlorine dioxide. Residual chlorine and chlorine dioxide levels are currently 0.1 mg/L. The residual chlorine value is anticipated to remain the same but the permitted residual chlorine dioxide level will drop to 0.05 mg/L.

Based on the 1981-84 data in Table 3, the Zurich waterworks could not meet all the proposed regulations with current treatment.

4.1.6 Future Direction

The future direction can be inferred by examination of work done with an experimental facility at Letten which began operation in 1983. This facility is treating water for artifical groundwater infiltration. The source is Limmat river water which is effectively Lake Zurich surface water. Several alternatives were investigated for pretreatment and the optimum treatment was identified as (Figure 3):

Pre-oxidation with 2.5 mg/L ozone

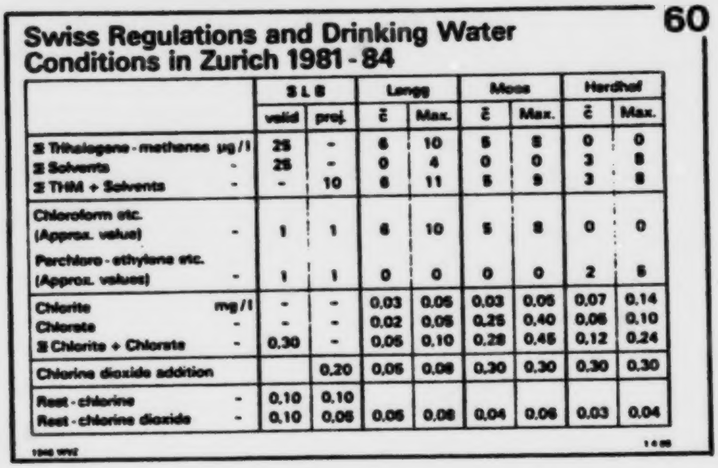
Microflocculation

Dual media filtration

Intermediate oxidation with 1.1 mg/L ozone

(Dual media?) activated carbon filtration (biological)

Chlorine dioxide (distribution system residual).



Swiss Regulations and Drinking Water Conditions in Zurich 1981 - 84

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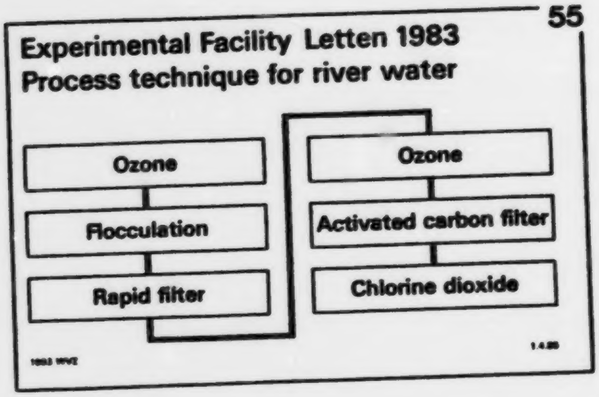
		S L B		Lengg		Moos		Hardhof	
		valid	proj.	c̄	Max.	c̄	Max.	c̄	Max.
Σ Trihalogene - methanes	µg / l	25	-	6	10	5	8	0	0
Σ Solvents	-	25	-	0	4	0	0	3	8
Σ THM + Solvents	-	-	10	6	11	5	9	3	8
Chloroform etc. (Approx. value)	-	1	1	6	10	5	8	0	0
Perchloro - ethylene etc. (Approx. values)	-	1	1	0	0	0	0	2	5
Chlorite	mg / l	-	-	0,03	0,05	0,03	0,05	0,07	0.14
Chlorate	-	-	-	0,02	0,05	0,25	0,40	0,06	0.10
Σ Chlorite + Chlorate	-	0,30	-	0,05	0.10	0,28	0,45	0,12	0.24
Chlorine dioxide addition			0,20	0,05	0,08	0,30	0,30	0,30	0,30
Rest - chlorine	-	0,10	0,10						
Rest - chlorine dioxide	-	0,10	0,05	0,05	0,08	0,04	0,06	0,03	0,04

Table 3 Drinking Water Regulations for Selected Parameters in Switzerland.

Source: Schalekamp (1986)



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Experimental Facility Letten 1983
Process technique for river water

Figure 3 Process Flowsheet for Letten Pilot Plant, Zurich

Source: Schalekamp (1986)

Ozone was selected over chlorine because it produced a greater reduction in the spectral absorption coefficient (UV extinction at 254 nm).

A key feature in Zurich treatment and in much of advanced European treatment is the use of multi-step oxidation. Schalekamp (1986) discusses the relative merits of each oxidant for each stage:

1. For pre-oxidation chlorine alone is not recommended because it produces unacceptable levels of trihalomethanes. Similarly pre-oxidation with chlorine dioxide alone is not recommended because it produces undesirable amounts of chlorite and chlorate. A mixture of the two has been found to minimize both types of reaction products. However, Schalekamp states that "ozone is by far the best since it produces no harmful by-products whatsoever provided that the raw water contains no bromine". This statement could be questioned as it obviously does not take into account possible harmful reaction products other than trihalomethanes from interaction between ozone and organics.
2. For intermediate oxidation only ozone is recommended since it facilities biological activity in subsequent slow or GAC filters. This produces in the Zurich situation filter run times which are 4 to 10 times longer than would be possible without the ozone.
3. For the final oxidation step to provide a distribution system residual, chlorine dioxide is in Schalekamp's opinion significantly better than chlorine. Chlorine imparts a taste to the water while chlorine dioxide is mainly degassed at the consumer's tap. It thus produces a smell but the concentration in the water is low and the resulting water has a pleasing taste. Ozone is not recommended as the final oxidant because of bacterial regrowth in the distribution system.

It is planned to convert both of the lake water plants to ozone for pre and intermediate oxidation in the relatively near future. The existing facilities need replacement and ozone will be suitable as an oxidizing agent, for protection against the shell larvae and to promote biological activity. The Moos and Lengg plants will then have pre- and intermediate oxidation with ozone, slow filtration directly following rapid filtration, intermediate ozonation followed by a biological activated carbon step and chlorine dioxide for distribution system protection (Figure 4). The maximum dosage of chlorine dioxide for distribution system protection is 0.05 mg/L which produces a residual of 0.01 to 0.03 mg/L. This is current practice and is expected to be continued. It is planned to add additional chlorine dioxide in the distribution system reservoirs if the maximum allowable dosages should be reduced in the future or if the danger of bacterial regrowth exists.

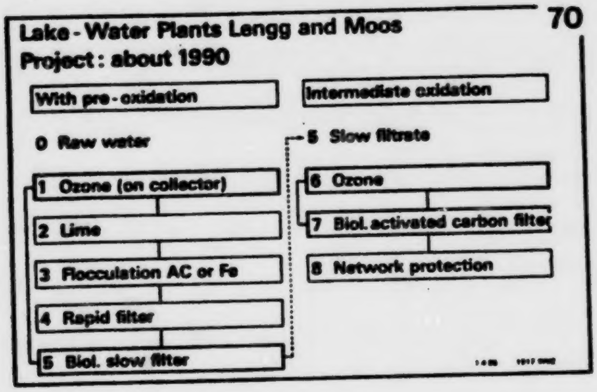
4.1.7 Costs

Schalekamp (1986) provides the following costs for the various oxidizing agents in Zurich. The first set of costs are based on 1.5 mg/L per oxidant and a flow of 36×10^6 m^3/year. The costs per m^3 are chlorine: 0.4¢ (Swiss Franc cents), chlorine dioxide 1.8¢, chlorine (1.0 mg/L) plus chorine dioxide (0.5 mg/L) 1.1¢, ozone 2.1¢.

The operating and capital costs (per m^3) for oxidation and subsequent carbon filtration, taking into account the longer flow time through biological filters are as follows:

chlorine	4.4¢ (Swiss Franc cents)
chlorine dioxide	5.8¢
chlorine/chlorine dioxide	5.1¢
ozone	4.1¢

Under these conditions ozone is then the cheapest oxidant.



Lake - Water Plants Lengg and Moos
Project : about 1990

70

With pre - oxidation

0 Raw water

1 Ozone (on collector)

2 Lime

3 Flocculation AC or Fe

4 Rapid filter

5 Biol. slow filter

Intermediate oxidation

5 Slow filtrate

6 Ozone

7 Biol. activated carbon filter

8 Network protection

Figure 4 Proposed Process Flowsheet for Lengg and Moos Plants, Zurich

Source: Schalekamp (1986)

To provide a rough conversion of these costs to Canadian conditions one Swiss Franc is equivalent to 72¢ or 73¢ Canadian (Globe and Mail Report on Business, February 15, 1986 and May 29, 1986, respectively).

4.1.8 Implications for Edmonton

Because the raw water quality in Zurich is not directly comparable to that in Edmonton a detailed comparison of individual process steps is not warranted. However, more general conclusions can be drawn from the type of processes used:

1. Zurich is moving towards the use of ozone as the major oxidant, with chlorine dioxide used for distribution system protection. The ozone will be applied at several places in the treatment train.
2. Granular activated carbon is used to provide removal of organic substances. In combination with ozone, a biological treatment effect is obtained, greatly extending the life of the carbon.
3. Zurich carries out process development in anticipation of possible stricter regulations in the future. Extensive use is made of pilot scale studies in developing new processes.
4. The emphasis appears to be in obtaining the best possible treatment rather than simply on meeting the regulatory requirements. An example of this is the use of a mixture of chlorine and chlorine dioxide to reduce both trihalomethane and chlorite formation.

4.2 Langenau, West Germany

This plant provides part of the supply for the City of Stuttgart, West Germany.

4.2.1 Source of Supply

The source of supply for this plant is the Upper Danube (Donau). As shown in Figure 5 the Danube rises near the Black Forest in West Germany and flows eastward across southern Germany before turning southward to flow through Austria, Hungary and other countries to the Black Sea. In the region of Langenau the Danube is a moderately sized river with an average annual flow of 140 m^3/sec. There is no chemical industry upstream, however there are two significant waste inputs: a cellulose factory 40 km upstream and the sewage plant of the City of Ulm (280,000 population equivalents) 20 km upstream.

Major parameters for raw water quality are given in Table 4. More detailed information on raw and finished water quality is given in Appendix 3.

Table 4 shows a raw water which is much closer to Edmonton's than is Zurich's. Levels of most parameters are higher than those for Zurich and reflect the greater variability expected in a river as opposed to a lake. Turbidity values and nutrient levels are many times higher than for Zurich, while organic carbon levels are approximately twice as great. The Danube water would be considered quite hard.

One interesting aspect of this plant is that it is one of only two in West Germany which take water directly from a river for treatment (Bernhardt, 1986). All other plants using a river source provide some form of storage or bank filtration prior to treatment.

4.2.2 Size of System

The total population served by the utility which operates the Langenau plant is approximately 2.5 million. This includes part of the population of Stuttgart and a number of smaller communities between the plant and the city. However, as discussed in the next section the Langenau plant was constructed relatively recently and provides only a small proportion of the

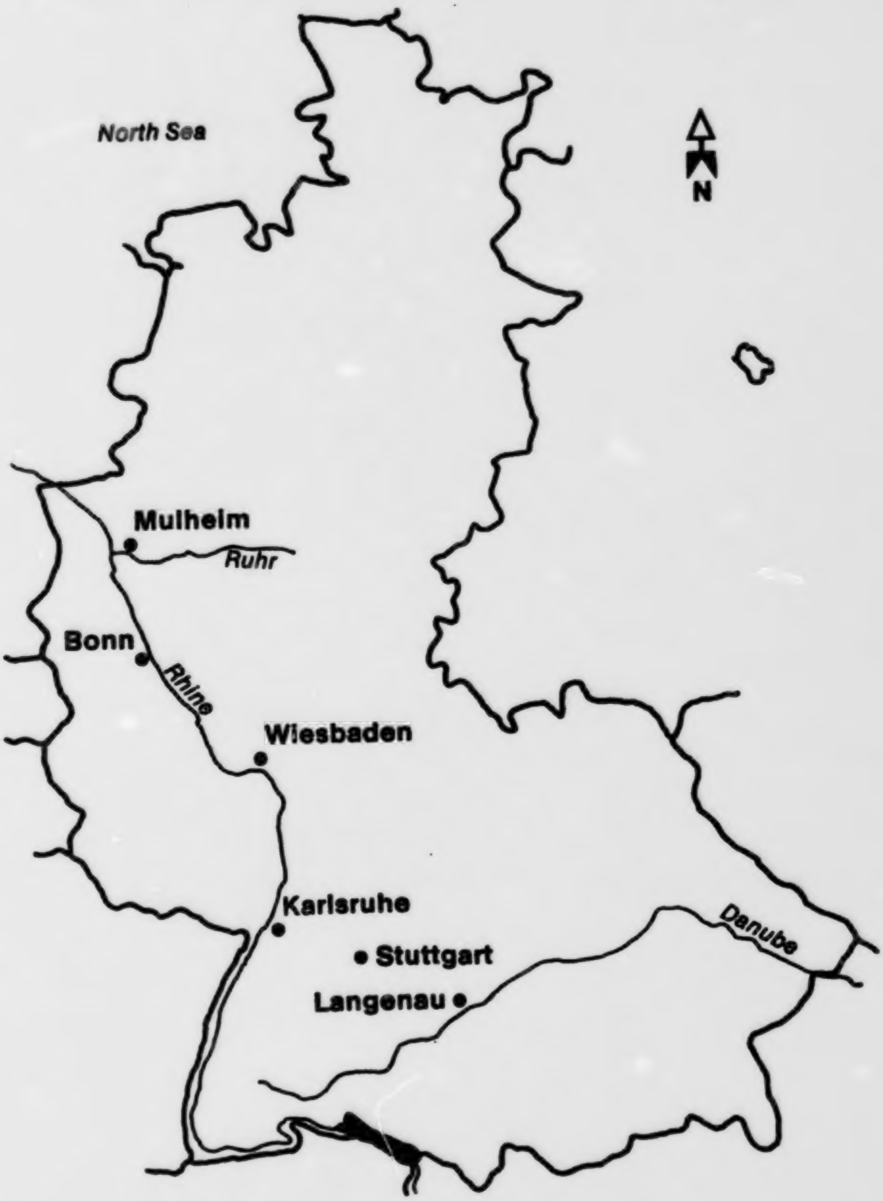


Figure 5 Map of West Germany

Table 4. Selected Raw Water Quality Parameters for the Danube River at Langenau, West Germany (1985 data).

		Reported Values		
Parameter	Units	Average	Minimum	Maximum
Temperature	°C	10.4	5.7	16.3
Turbidity	FTU	8.0	1.0	54
pH		7.50	7.21	7.72
Conductivity	µS/cm (20°)	470	360	560
Total hardness	°dH[a]	15.0	11.2	16.8
	mg/L $CaCO_3$	268	200	300
Chloride	mg/L	23	16	30
Dissolved oxygen	mg/L	6.6	3.7	9.8
NH_4^+	mg/L	0.20	0.05	0.45
Orthophosphate	µg/L	360	77	543
DOC	mg/L	2.7	1.8	3.2

[a] German degrees of hardness 1 °dH = 17.86 mg $CaCO_3$/L

total production. For 1979 the annual plant production was 12 x 10^6 m^3 out of a total production of 80 x 10^6 m^3, or approximately 15% (Landeswasserversorgung, undated #2). The maximum plant capacity is 2.3 m^3/s or 200,000 m^3/d. Werner (1983) reported that plant production varied from a minimum of 17,000 m^3/d up to the maximum capacity.

4.2.3 Historical Development

The Landeswasserversorgung (State Water Supply) was founded in 1912. From 1912 to 1917 the first groundwater production plants near the Danube were installed and a transmission line approximately 100 km long was constructed to carry this water to Stuttgart.

Additional groundwater works were brought into service at various times up to 1955. In 1965, the Zweckverband (local administrative union) was formed, taking over the facilities and provision of water previously performed by the various municipalities.

The Langenau treatment plant was constructed during the period 1967 to 1973 along with a third transmission line to Stuttgart. The Langenau waterworks was the first one in the system to treat surface water and the goal of this treatment was and remains to produce drinking water equal in quality to the high quality groundwater obtained in this region.

In 1981 a compact flocculation plant was brought into operation ahead of the existing process to improve treatment. Since that time pilot scale studies on softening have been conducted at the Langenau plant.

4.2.4. Scope of Discussion

This report will discuss only the surface water treatment at Langenau. The emphasis in the discussion will be on flocculation and filtration, and especially the compact flocculation plant. This is a unique feature of the Langenau waterworks and has been studied and reported on in detail. Of particular importance and interest are the philosophy and approach: the process was developed because the utility's goal was to be able to respond to all raw water conditions without a reduction in either finished water quality or plant throughput. Prior to construction the process was evaluated and optimized through extensive laboratory and pilot scale studies lasting several years.

4.2.5 Process Prior to 1981

1. Description

The treatment process at Langenau prior to 1981 as shown in Figure 6 is described by Bernhardt (1985). The first treatment step consisted of a rapid mix unit in which oxidant and iron sulfate were added. The iron dosage was approximately 6 mg/L Fe^{3+}. Chlorine was originally added as the oxidant but this has since been discontinued. Flocculation and settling took place in a solids contact unit (Accelater) to which lime was at least sometimes added, although this is not discussed in any of the reference material. A polyacrylamide in a dosage of 0.25 to 0.4 mg/L was also added. The vertical rise rate in the Accelater was 4.7 m/h.

Following this ozone in a dosage of 0.8 to 1.2 mg/L was added in the main oxidation step. Secondary flocculation was carried out by the addition of 0.2 to 0.8 mg/L Fe^{3+} to each filter. The dosage was decreased as filter run time increased.

Filtration was provided by ten gravity dual media filters with the following media specifications: 1.1 m anthracite (1.6 to 2.5 mm diameter), 1.2 m sand (0.6 to 1.0 mm diameter). These were installed directly above activated carbon filters with a 1.5 m deep bed and a filtration velocity of 15 m/h. The carbon type and size were not given.

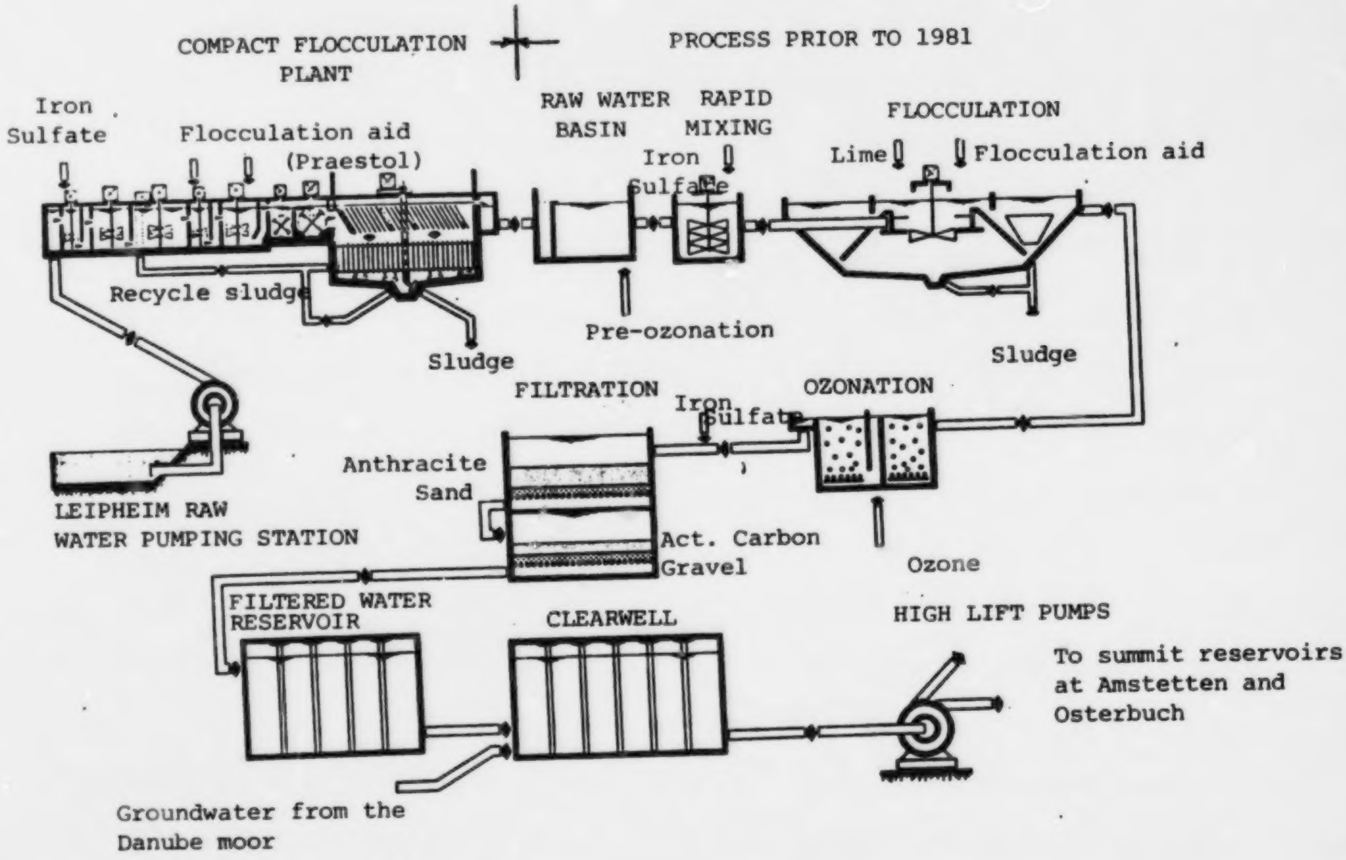


Figure 6. Process Schematic for Langenau Waterworks

Source: Wölfel et al (1982)

Filtered water was mixed with groundwater prior to distribution. Post chlorination was used to provide a distribution system residual.

2. Performance

Bernhardt (1985), with particular reference to flocculation and filtration, reports that the plant produced an excellent quality water. Residual iron concentrations were less than 0.005 mg/L and residual turbidities were less than 0.05 FTU. This was possible, according to Bernhardt, because of the following critical process steps which were new at that time: turbulent premixing ahead of the Accelater; a solids contact step with variable and high energy input; preozonation combined with secondary flocculation; and dual media filtration followed by GAC.

The use of the solids contact unit and secondary flocculation was shown to be very important for filter run time. Pilot plant data showed that without a solids contact unit filter run times varied from seven to 18 hours depending on the raw water turbidity. This was considered to be unacceptable, because even with high turbidity loadings filter run times should be approximately 100 hours according to Bernhardt (1985). With the solids contact units and secondary flocculation filter run times were increased to three days without ozone and five days with ozone.

The importance of secondary dosing of iron in the filter feed was shown clearly in pilot plant studies. The particles of concern range in size from <1 to 2 microns. Table 5 shows the results of a run in which the dosing of iron in the filter influent was periodically interrupted. Filter effluent turbidities approximately doubled and residual iron concentrations more than doubled when the iron feed was discontinued. This effect was apparently noticeable approximately 20 minutes after discontinuation of the iron dosage.

The results in Table 5 were obtained without ozone addition. It was

Table 5. Leipheim* Pilot Plant (1967). Filter Run with Changing Secondary Flocculant Dosage (without Ozone Addition).

Secondary Dose (mg/L Fe^{3+})	1	0	1	0	1	0	1
Filtrate Turbidity (FTU)	0.10	0.23	0.10	0.17	0.10	0.18	0.09
Filtrate Residual Iron Concentration (mg/L Fe^{3+})	0.014	0.063	0.012	0.027	0.008	0.024	0.005

* Location of the intake on the Danube.

After Bernhardt (1985)

shown, however that ozone addition prior to the secondary flocculation considerably aided the removal of hard-to-filter material. At low temperatures especially this material was hardly removed in the top layer of the filter and only in the sand. This combination of ozone with secondary flocculation first demonstrated in a full scale operation the possibility of achieving such low turbidities (0.05 FTU) and iron concentrations (0.005 mg/L) (Table 6). Thus the process was capable of producing an excellent filtrate under normal conditions. The process also allowed the residual polymer concentration in the filtrate to be "neglectably small" (less than 1 µg/L measured according to the method of Weindel and Sontheimer (1971)).

Bernhardt (1985) notes that the success of secondary flocculation depends on sufficient destabilization of the small particles in the initial rapid mix zone. Secondary flocculation in the filters is thus not intended to provide destabilization of colloidal material but rather to flocculate fine destabilized particles which have not had an opportunity to settle during the sedimentation step. Bernhardt states that this appreciation of the true role of secondary flocculation is frequently overlooked in waterworks.

4.2.6 Compact Flocculation Plant

1. Need, Concept and Development

In the years after placing the Langenau plant into service it was found that poor water quality in the Danube was not always satisfactorily handled by the plant. The river was subject to very large and sudden changes in the content of inorganic suspended material and high concentrations could last for several weeks. Levels of suspended solids could increase to over 200 mg/L

Table 6. Influence of Secondary Flocculation with Iron (III) Salts Immediately Before Filtration (Full Scale Data).

a. Unoptimized Secondary Flocculation (Break-in Phase, 1973).

Turbidity (FTU)		
Raw Water	Accelator Effluent	Filtrate
2.75	1.2	0.25
3.4	1.3	0.26
2.2	1.25	0.16
3.3	1.65	0.30

b. Optimized Secondary Flocculation (Optimized dose added in the feed of individual filters).

Turbidity (FTU)		
Raw Water	Accelator Effluent	Filtrate
2.8	0.65	0.05
2.6	0.40	0.06
2.4	0.55	0.06
2.7	0.39	0.05
2.8	0.45	0.05
2.5	0.6	0.05

Throughput in all cases: 400 L/s in each Accelator.

After Bernhardt (1985)

during periods of high water and values as high as 1,500 mg/L were measured. Some of this material was finely dispersed and hard to remove.

In addition, the sludge recycle step in the Accelators did not work properly during extreme high water conditions with high concentrations of inorganic suspended solids. The recycle sludge apparently became too heavy and lay on the bottom of the Accelator. Plant throughput had to be reduced, and this could have caused a problem if it had coincided with a period of high water demand.

Problems were also experienced under low water conditions: high levels of organics from wastewaters, especially the upstream cellulose industry, could result in considerable impairment to the flocculation process. Normal DOC levels of 2.5 to 4 mg/L could rise as high as 7 mg/L under low water conditions and might last for several weeks.

Water temperature in the Danube could be as low as 0.5°C in winter and this could impair flocculation.

A critical operational problem was encountered in the summer of 1976. Several months of extreme low flows coupled with high concentrations of colloids and dissolved organic substances impaired flocculation such that the dual media filters and GAC were not able to operate at their normal throughput of 2,000 L/s.

Bernhardt (1985) states that these factors "show clearly how important it is to design a plant producing drinking water from a river for the maximum concentrations of the various parameters important for finished water quality which are controllable through flocculation and optimal floc removal".

On the basis of the foregoing considerations and also the necessity of raising the maximum plant throughput to 2,300 L/s an additional pre-treatment step (compact flocculation plant) was developed and built. This step was added to the beginning of the existing process and the Accelators were retained as bio-reactors for oxidation of ammonia and organics.

The primary requirement of the new process step was to allow the complete plant to always operate in an assured and stable manner. The specific goals of the compact flocculation plant were (Wölfel et al, 1982):

1. To remove completely the difficult-to-flocculate organic colloids encountered during extreme low water flows in the Danube.
2. To remove, virtually quantitatively, the gram per litre quantities of inorganic suspended solids experienced under high water conditions, without heavy sludge settling out in the plant.
3. To remove raw water contaminants through flocculation and solids separation without the use of a disinfectant and especially without the use of chlorine. In addition the sludge deposited in the plant should not undergo decomposition. This requirement was made in connection with chlorination in the following Accelator for chemical ammonia oxidation. It is not clear whether the investigators wished to avoid the use of chlorine for chemical oxidation or only wanted to avoid the formation of organic chlorine compounds if chlorination for ammonia oxidation were required.
4. To produce a residual suspended solids concentration always less than 1 mg/L in the effluent from the compact flocculation step, independently of raw water quality. This requirement was set, among other reasons, to allow a virtually chlorine-free operation of the entire plant, permitting the use of the Accelators as bio-reactors. For the bio-reactors a high sludge age is necessary. This requires an especially small suspended solids concentration in the influent and a minimal sludge withdrawal.

Thorough consideration and calculations showed that a pretreatment step of the type desired could be constructed efficiently as a flocculation plant with a following floc separation step and that such a project could also be constructed for a reasonable capital cost. For the given demands a type of solids contact process seemed especially suited. The theoretical basis of such processes had already been established through previous investigations in the laboratories of the Landeswasserversorgung (Werner, 1969; Wurster and Werner, 1971).

These previous bench scale studies had shown that a solids contact process, which is the process of choice for treating high turbidity waters (Wölfel et al., 1982), can be divided into five basic phases:

Phase I - addition of flocculating agent, destabilization of colloids and formation of micro-flocs
Phase II - contact of the micro-flocs with the recycled sludge
Phase III - addition of flocculation aid
Phase IV - development of removable large flocs
Phase V - separation of these large flocs through sedimentation

The consequence of this detailed understanding is that each phase will have an optimum contact time and energy input, and so for true process optimization the steps should be separated. Testing and development of the process was therefore carried out with a pilot plant in which the individual process steps were accomplished in separate compartments (Figure 7). Both energy input and contact time in each compartment could be controlled and optimized. A Lamella separator mounted on top of a thickener was used for solids separation.

Pilot scale investigation and optimization of the process lasted two years; details are provided by Wölfel, et al (1982). Residual iron following

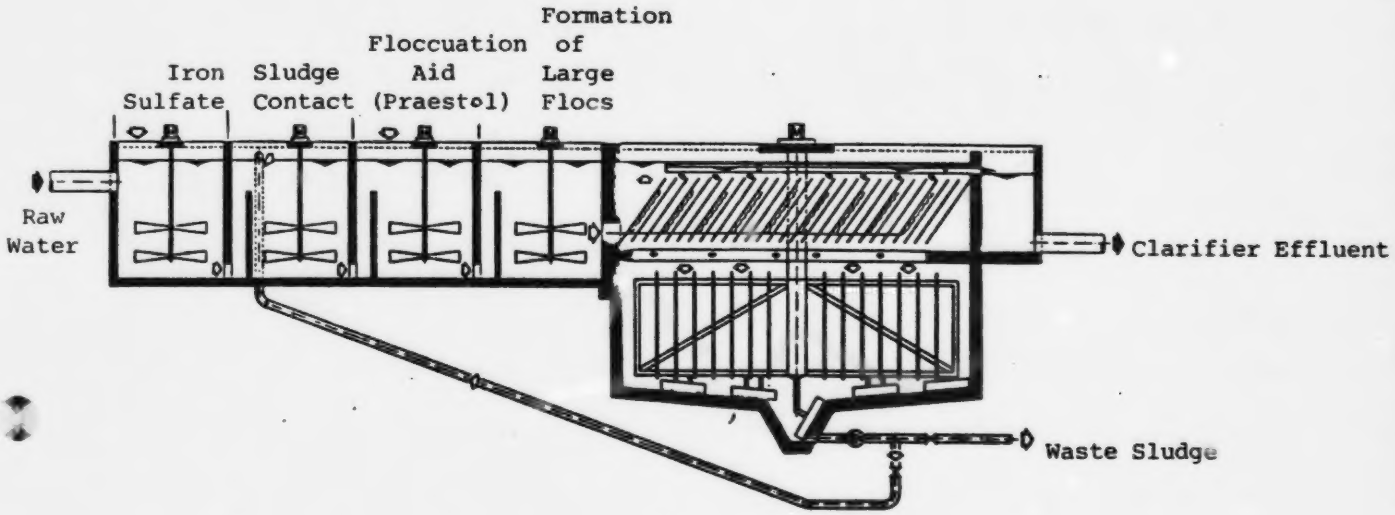


Figure 7. Process Schematic of Compact Flocculation Pilot Plant, Langenau

Source: Wölfel et al (1982)

sedimentation was used as the control parameter and Danube water was sometimes spiked with both organic material and turbidity to simulate poor water conditions. In addition to defining optimum contact times and energy inputs the investigation also evaluated different impeller types for agitation. As a result of the investigation several of the reaction chambers were subdivided, e.g. chamber I was divided into Ia and Ib, etc.

Example pilot scale data in Figure 8 show the effect of different influent turbidity and organic levels on the residual iron concentration obtainable and the minimum energy input necessary for process phase III (addition of floccant aid).

The full scale plant took two years to construct and was placed in operation in June 1981. It consists of two parallel trains, each designed for 750 L/s (2,700 m^3/h). The cost of construction was approximately 11 million deutsche Marks (DM). The approximate equivalent in Canadian funds would be $5.5 million.

Technical details of the plant are provided in Table 7.

2. Performance

Tables 8 and 9 show the improved efficiency of the new plant compared to the old one in dealing with incidents of poor raw water quality. In June 1983 the turbidity rose in a few hours from 4-5 FTU to 150 FTU (Table 8). At the same time the DOC rose from 1.9 to 3.5 mg/L. As an indication of the extremely high efficiency and adaptability of the process, the effluent turbidity from the compact flocculation plant rose only from 0.24 to 0.95 FTU during this time.

This effluent turbidity increase was not only due to the rise in raw water turbidity. At the same time, presumably because of demand, the plant throughput was increased from 450 to 750 L/s (full capacity). At this time

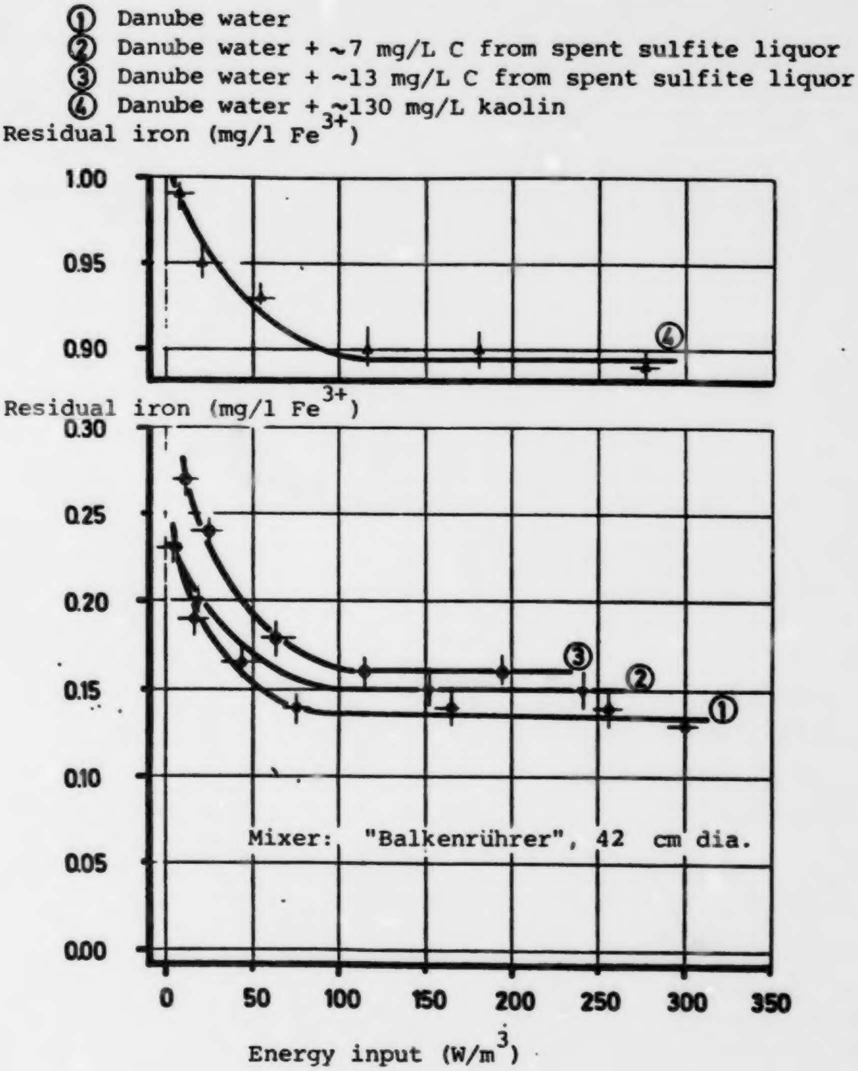


Figure 8. Influence of Energy Input in Phase III on Supernatant Residual Iron Concentrations Following Sedimentation (Langenau Pilot Plant)

Source: Wölfel et al (1982)

Table 7. Technical Features of the Compact Flocculation Plant[a]

Reaction Times and Energy Inputs

Chamber	Phase	Volume (m^3)	Reaction Time[b] (min)	Energy Input			
				W/m^3		G Value[c] (s^{-1})	
				Avg.	Max.	Avg.	Max.
Ia	I	14	0.3	200	600	390	680
Ib	I	70	1.5	60	225	215	420
II	II	259	5.8	40	160	175	350
IIIa	III	24	0.5	100	370	280	530
IIIb	III	55	1.2	100	370	280	530
IVa	IV	145	3.2	1	5	28	62
IVb	IV	208	0.6	0.02	0.5	4	20

[a] Values given are for 1 of 2 parallel streams.
[b] For Q = 750 L/S (2,700 m^3/h)
[c] for 10°C

Thickener volume: 1,572 m^3
Total reaction time for flocculation: approx. 17 min[b]
Total reaction time including settling: approx. 20 min.

Dosages

Iron (III) sulfate	approx. 0.1 - 0.14 mmol/L Fe^{3+} (6 - 8 mg/L Fe^{3+})
Polyacrylamide (Praestol)	0.2 - 0.25 mg/L
Recycle sludge (compared to raw water throughput)	1 - 2%

Suspended Solids Content

in effluent (up to approx. 400 L/s)	< 0.5 mg/L TS
in contact sludge	2% TS
in wasted sludge (depending on raw water solids content)	approx. 7-14% TS

Mixer energy requirements

per m^3 treated water	approx. 0.01 kWh

After Wölfel et al (1982)

Table 8. Effect of a High Water Discharge in the Danube with High Turbidity and DOC Concentrations, with Different Water Throughputs on the Effluent of the Compact Flocculation Plant and Dual Media Filters.

Date (1983)	Danube Discharge (m^3/s)	Throughput (L/s) (each stream)	Raw Water Turbidity (FTU)	Raw Water DOC (mg/L)	Compact Flocculation Plant Doses Fe^{3+}* (mg/L)	Doses Praestol (mg/L)	Effluent Turbidity (FTU)	Effluent DOC (mg/L)	Energy Input (W/m^3) Ia	Ib	IIIa	IIIb	Filter Effluent Turbidity (FTU)
June 13	138	400	4.5	1.9	6	0.3	0.24	1.4	230	42	97	25	0.04
June 14	174	750	5.2	-	12	0.3	0.68**	-	230	42	97	25	0.04
June 15	394	600	148	-	12	0.3	0.95**	-	230	42	97	25	0.05
June 16	282	400	110	3.5	20	0.3	0.59	1.9	240	110	90	85	0.04
June 18	184	200	13	-	12	0.3	0.24	-	240	110	90	85	0.04
June 20	150	200	4.8	2.0	8	0.3	0.31	1.7	230	42	90	45	0.04

* Applied as Iron (III) sulfate with 10% Fe^{3+}.

** Turbidity consisted mainly of finely dispersed iron hydroxide, which was removed in the filter. Because the filtrate turbidity (0.04 FTU) did not initially increase, the energy input was not changed. An increase was made when the filtrate turbidity rose to 0.05 FTU.

Dosages in Bioreactor: 3 mg/L Fe^{3+}
0.2 mg/L Praestol (June 13-16)
0.15 mg/L Praestol (June 18-20)

After Bernhardt (1985)

(June 14 and 15) the energy input was unchanged. The filtrate turbidity rose by only 25% to 0.05 FTU, and it decreased again on June 16 after the energy input and iron dose had been increased. At this time the flow was also reduced again to 400 L/s. The effluent turbidity of the compact flocculation plant then returned to 0.59 FTU, the value before the rise in raw water turbidity. This effluent was achieved with a raw water turbidity of 110 FTU.

These results can be compared to an experience in February 1980, before the compact flocculation plant was brought into service (Table 9). A rise in the raw water turbidity from 9 to 80-96 FTU resulted in an Accelator effluent turbidity of approximately 1 FTU. While not that different than the value measured in 1983, it occurred despite an increase in the iron dose from 16 to 26 mg/L for several days. More importantly the turbidity of the filtrate rose from 0.04 to 0.07 FTU, a 75% increase from the relatively stable normal value. The plant throughput was also considerably lower than in 1983, remaining at 400 L/s. The one point to note in this comparison is that the 1980 incident occurred during the winter when flocculation may have been more difficult because of low temperature.

Bernhardt (1985) states that "from these examples it is clear that this newly developed system of energy-input-controlled flocculation with sedimentation and sludge contact represents an advance in flocculation technique".

The results show that the finished drinking water contains extremely low concentrations of turbidity and flocculation chemicals. The plant and especially the energy input are controlled on the basis of the filtrate turbidity and iron concentration. The goals are respectively <0.05 FTU and <0.01 mg/L Fe. An additional factor in the dosing of flocculating chemicals is the turbidity of the raw water and of the flocculation plant effluent.

Table 9. Effect of a High Water Discharge with High Turbidity on Accelator Effluent and Dual Media Filtrate Before Construction of the Compact Flocculation Plant.

Date (1980)		Danube Discharge (m^3/s)	Throughput (L/s)	Turbidity (FTU)			Dosages (mg/L)	
				Raw Water	Accelator Effluent	Filtrate	Fe^{3+}	Praestol
February	1	212	300	9.3	0.46	0.04	8	0.2
	2	244	300	21	0.75	0.06	16	0.2
	4	242	300	14	0.78	0.05	16	0.2
	5	461	300	24	0.71	0.05	16	0.2
	6	656	400	96	1.2	0.07	26	0.3
	7	536	400	78	0.89	0.07	26	0.3
	8	526	400	81	0.92	0.07	26	0.3
	9	423	400	37	0.54	0.06	20	0.25
	11	299	400	16	0.54	0.05	12	0.2
	12	296	400	11	0.36	0.05	12	0.2

After Bernhardt (1985)

Table 10 provides a comparison of operating parameters for the compact flocculation plant with those of the Accelator when it was used for flocculation.

Operating experience with the plant has been good (Wölfel, 1985). No problems have been reported with algae or other growths on the plates of the Lamella separator, something which perhaps had been anticipated. Good ammonia removal is obtained in the bio-reactors even in winter. The plant is designed to handle rapidly fluctuating flows and according to Table 10 is able to be brought into service effectively immediately without problems.

During the writer's site visit in 1983 the compact flocculation pilot plant was being used in the same process configuration for softening. Wölfel (1985) reports that this has been successfully demonstrated.

Werner (1983) reported that the basic operating strategy when water quality deteriorates is to increase the energy input to selected process steps. There are set operating levels which are used in order to simplify operation. If the first increase in energy input does not improve the compact flocculation plant effluent the operator is instructed to increase the energy to the next level.

4.2.7 Additional Treatment Steps

The discussion up to this point has focused largely on the compact flocculation step. A brief discussion of the operation of the other process steps is now provided, based on information obtained by the writer during a site visit in 1983 (Werner, 1983).

Referring to Figure 6 it is evident that the treatment consists of seven main steps:

1. The compact flocculation plant - removal of trihalomethane precursors is 50% in this step.

Table 10. Comparison of Operating Parameters for Accelators[a] and Compact Flocculation Plant.

	Accelator	Compact Flocculation Plant
Surface loading ($m^3/m^2.h$ or m/h)	4.7	36
Time for flocculation and sedimentation (min)	75	20
Startup time on taking into service (without contact sludge)(h)[b]	6	0.25
Restart time after standing still (with contact sludge)(h)[b]	3	0
Concentration of wasted sludge (%TS)	0.5	7-14

[a] Before introduction of compact flocculation plant

[b] To produce normal effluent turbidity.

After Wölfel (1985)

2. First ozone addition (0.2 to 0.4 mg/L)
3. Bioreactors - ammonia removal and precursor removal (10 to 20%) take place in this step.
4. Main ozone addition (0.6 to 1.6 mg/L) - this is the first disinfection step. In addition it provides oxidation of trihalomethane precursors and of other oxidizable organics. It also contributes to effective dual media filtration.
5. Filtration - an iron dosage of 0.2 to 0.5 mg/L is added to the influent of each filter for secondary flocculation. The normal filtration rate of 15 m/h can be increased to a maximum value of 20-22 m/h. Backwash water for the filters is 0.3% of throughput and backwashing normally takes place once or twice per week.
6. Granular activated carbon - both adsorption and biological activity take place in this step. Both ammonia and the rest of the trihalomethane precursors are removed. A removal of general organics (presumably expressed as TOC) of 30-40% is achieved. "Harmful" organics are reportedly removed to below detection limits.
7. Disinfection with chlorine - A dosage of 0.4 to 0.5 mg/L is added. Because of the extremely high quality of the finished water this is able to provide a detectable residual in Stuttgart approximately 80 km away.

The treated water is essentially equivalent in quality to the ground water with which it is blended prior to transmission and distribution. Haloform levels in the finished water are <5 μg/L.

Detailed data on treated water quality are provided in Appendix 3.

4.2.8 Costs

Up-to-date cost information was requested but not obtained. However an average income from water sales of 46.6 Pfennig/m^3 was reported for 1976 (Landeswasserversorgung, undated #1). This was before the introduction of the compact flocculation plant. As a very rough approximation a value of 2 Pfennig per Canadian cent can be used, giving an approximate selling price at that time of 25¢/m^3.

4.2.9 Implications for Edmonton

The experience at Langenau particularly with respect to the compact flocculation plant demonstrates what can be achieved given a commitment to process optimization. Excellent treatment is obtained with a process which is basically physical rather than chemical and is both flexible and simple. It offers the possibility of perhaps avoiding the necessity of granular activated carbon or certainly of conserving its capacity. No oxidant is added early in the process, thus reducing undesirable by-product formation. Although a polymer is added during treatment the residuals are very low. Of particular importance for Edmonton is the fact that the excellent turbidities achieved should provide a high degree of protection against Giardia by physical means alone.

A beneficial effect in the distribution system should be achievable with this level of treatment as a lower chloramine dosage would be required.

The possibility of operating the process with aluminum rather than iron as coagulant should also be assessed if this has not already been done at Langenau. Since solids contact clarifiers already exist at the E.L. Smith plant, as a minimum step the improvement of their performance by a separate high energy rapid mix step should be investigated. The possibility of using the compact flocculation process for softening is also of interest.

Although the whole issue of the use of biological treatment needs to be investigated and decided by Canadian regulatory authorities, biological processes are certainly worthy of attention.

4.3 Méry-sur-Oise, France

The Méry-sur-Oise waterworks is one of the large drinking water plants serving the Paris suburbs. As with other plants belonging to the Water Authority of the Paris Suburbs it is managed by the Compagnie Générale des Eaux. The plant is located on the banks of the Oise River about 15 km upstream from where it converges with the Seine (Figure 9).

Unless otherwise indicated information on this plant is taken from published plant descriptions obtained during a site visit in 1984. No authorship is given but the information is dated 1981 and 1983.

4.3.1 Source of Supply

The plant takes it water from the Oise River which is the largest tributary of the middle Seine. Draining from northeast France into the centre of the Paris basin, the Oise has a catchment area covering nearly 17,000 km^2. The Aisne River is the main tributary of the Oise and drains slightly less than half of this area. Flow in the Oise is steady and abundant and the plant is not faced with any major problems with regard to quantity.

Approximately 1.5 million people live in the Oise-Aisne basin, where considerable economic activity occurs, accounting for 5% of all such activity in France. There are many and varied industries, certain of which (e.g. paper mills, sugar refineries, canneries, chemical and dye plants) produce contaminants particularly deleterious to water quality. The residual pollutant load following waste treatment is estimated at 1.5 million population equivalents. The Oise is connected by a canal system to the Somme, the Sambre and beyond into Belgium and the Netherlands, resulting in considerable shipping traffic. Spills of contaminants are of particular

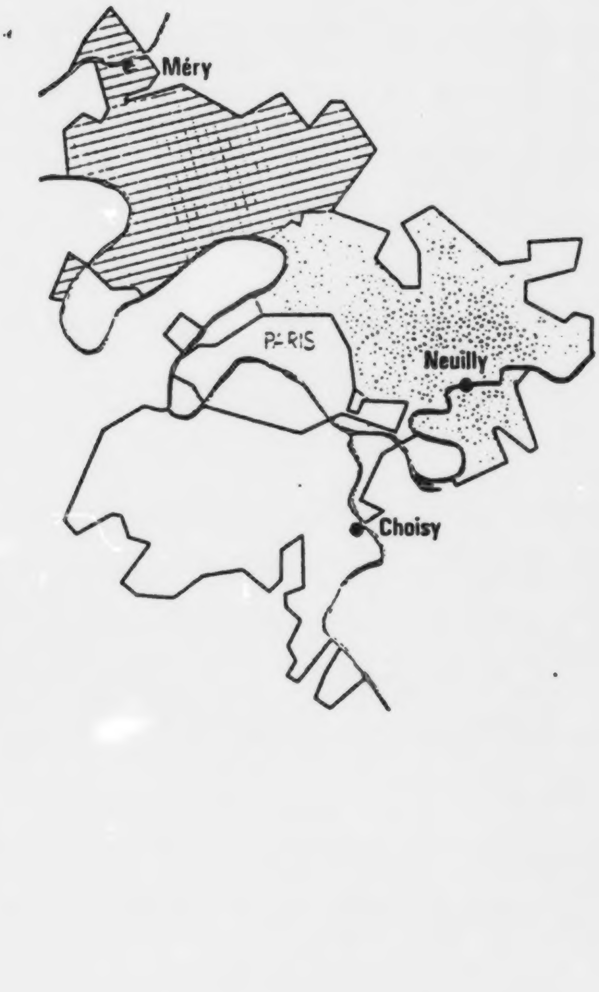


Figure 9. General location of the Méry-sur-Oise plant.

concern for water quality.

Major raw water quality parameters for the Oise River are summarized in Table 11. A more detailed characterization is provided in Appendix 3. As is evident from Table 11 the pH is in the alkaline range. The water has a high level of hardness, and adequate dissolved oxygen levels. Ammonia levels can range up to high values. Organic matter is reported primarily in terms of potassium permanganate values, but limited TOC data show approximately the same level. The river is reported to show less bacterial contamination than the other rivers supplying the Paris suburbs (the Seine and the Marne) but is heavily contaminated with inorganic and organic compounds.

4.3.2 Size of System

The Méry-sur-Oise plant serves a total population of 630,000 in 40 different communities. It has an annual production of 42 million m^3 with a maximum daily production of 270,000 m^3. Information from 1983 indicated that capacity was being expanded to 360,000 m^3 per day.

4.3.3 Historical Development

A water treatment plant was first installed at the Méry site in 1911. Its production was 20,000 m^3 per day and the treatment consisted of slow sand filtration followed by disinfection with a chlorine-based product.

Changes to the treatment process took place over years in response to increasing population growth in the Paris suburbs and deteriorating water quality as a result of this urbanization and industrial development in the watershed.

The present treatment plant was constructed in successive phases beginning in 1962. A rapid filtration treatment process was placed into operation and ozone disinfection was incorporated in 1965. The latest phase, realized in 1980, added raw water storage, pre-ozonation, filtration through

Table 11. Selected Raw Water Quality Parameters for the Oise River

Parameter	Units	Reported Values		
		Average	Minimum	Maximum
Temperature[a]	°C	12.9	5.1	20.3
Turbidity[b]	c	217	-	-
Suspended solids[b]	mg/L	45.5	-	-
pH		7.97	7.6	8.5
Resistivity	ohm-cm (@ 20°C)	1637	1236	2224
Total hardness	French degrees[d]	29	22.4	35.4
	mg/L $CaCO_3$	290	224	354
Dissolved oxygen	mg/L	9.1	6.1	12.8
NH_4^+-N	mg/L	0.44	0.02	1.6
Organic matter ($KMnO_4$)	mg/L	4.35	1.10	10.9
TOC[e]	mg/L	4.60	-	-

[a] Stored water, data for Sept. - Dec. 1980

[b] Data for Sept. - Dec. 1980

[c] Units not readable

[d] One French degree of hardness = 10 mg/L $CaCO_3$

[e] Data for Sept. - Dec. 1980 ($KMnO_4$ value for the same period was 3.87 mg/L)

Source: Plant documentation dated 1981.

granular activated carbon and the incorporation of biological treatment steps.

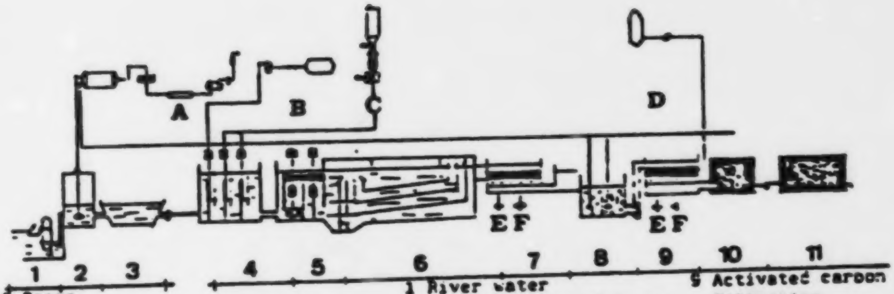
4.3.4 Current Process

A process schematic is given in Figure 10. The main treatment steps consist of: pre-ozonation and storage; addition of chemical reactants such as coagulants; flocculation, sedimentation and sand filtration; ozonation to promote biological activity followed by filtration through granular activated carbon; post-ozonation; and disinfection for distribution system protection. Each of the treatment steps is described in more detail below.

1. Pre-ozonation and storage

Raw water storage is provided as a protection against spills and accidental contamination of the river. This storage provides between 1.5 and 3 days supply depending on the production rate of the plant. The total volume of the storage basin is 390,000 m^3 with an active volume of 370,000 m^3. The surface area of the water is 5.3 hectares and the average depth is 9 m. Construction began in 1977 and took 3 years. This was apparently the first use of raw water storage in conjunction with surface water treatment in France.

The inlet to the basin was designed to avoid creation of stagnant zones. Cavelier et al. (undated) reported that the basin was essentially designed as a settling tank with high speed injection of the water to prevent stratification. Billen et al. (1984) reported that most inputs were at night, and that actual retention time was four days. Raw water is normally conveyed by gravity from the basin to the treatment plant. If a spill in the river required the intake to be shut down, stored water in the reservoir would be pumped to the treatment plant.



A Ozone
B Aluminium polychloride (W.A.C.)
C Powdered activated carbon
D Sodium hypochlorite

1 River water
2 Preozonation 0.5–1 ppm
3 Storage
4-5-6 Coagulation–Flocculation – settling
7 Sand filtration
8 Second stage ozonation 1.5–3 ppm
9 Activated carbon filtration
10 Post ozonation 0.4 ppm
11 Drinking water

Figure 10. Process schematic for Méry-sur-Oise plant

Source: Agbalika et al. (1985)

The natural treatment effect obtained by storage is enhanced by application of ozone prior to the entry of water into the basin. A typical dosage is 0.5-1 mg/L with a four minute contact time (Agbalika et al., 1985).

Pilot scale studies prior to construction of the full scale basin indicated that pre-ozonation and 3 days storage would reduce ammonia levels by 60%, pathogens by 90% and turbidity by 50%. For the full-scale basin Billen, et al. (1984) reported that on an annual basis organic solids were reduced by 58%, particulate organic carbon by 55%, and plankton biomass by 40% including growth inside the basin. These removals were limited by the circulation of water caused by the daily inputs. The resulting slight increase in turbidity regulated primary production despite the presence of nutrients. Daytime destratification of the water also prevented the build up of an anoxic zone. Under normal conditions the reservoir behaved like a sluggish river, however after two years in service it showed a certain "drift" because of the accumulation of sediments and changes in the quality of the raw water.

Table 12 provides data on the effect of the reservoir during the period April to October 1982. Some reductions in all measured parameters were observed.

2. Addition of Chemical Reactants

Agbalika et al. (1985) report that aluminum polychloride is used as the coagulant at doses varying from 15 to 20 mg/L. The coagulant is a substance produced in France and known as WAC (water aluminum chloride). The dosage was reported for a study on virus removal carried out in 1982 and is presumed to represent normal practice.

There is also the capability of adding powdered activated carbon at this step in the treatment process but this is apparently not normally done.

Table 12 Effect of the Storage Reservoir at Méry-sur-Oise on Selected Water Quality Parameters

Parameter	Units	Range of Values	
		Raw River Water	Stored Water
Dissolved oxygen	mg/L	7.2 - 10.5	5.8 - 9.7
Turbidity	JTU	12 - 36	6 - 12
pH		8 - 8.1	7.8
COD	mg/L	2.7 - 4.6	1.3 - 3.8
NH_4	mg/L	0.36 - 0.57	0.24 - 0.50

After Agbalika et al. (1985)

If the need arises, additional chemicals such as ferrous sulphate, soda or potassium chloride can be added. As is evident in Figure 10, a compartmentalization of the chemical addition step is provided to avoid undesirable interferences among the chemicals added. The ferrous sulphate is added in case of contamination by hexavalent chromium and sodium hypochlorite is added in case of high concentrations of manganese.

In the information dated 1981 chlorine dioxide was shown being added in this step but this practice has since been discontinued.

3. Flocculation, sedimentation and sand filtration

The only detail provided for the flocculation step is that the time is 30 minutes. The Hazen speed (presumably settling velocity) in the sedimentation tanks is 1.1 m/h and a minimum sedimentation time of 2 hours is provided.

Sand filtration is provided by 12 units each having a surface area of 108 m^2. The sand bed is 1.4 m deep with a grain size of 1 mm. Further information such as effective size and uniformity coefficent was not available. Using a special flow regulator the flow is distributed evenly over the 12 filters at a rate previously applied to 18 filters. The filtration rate was therefore increased from 6 to 9 m/h. Agbalika et al. (1985) report a filtration rate varying from 3.6 to 5.1 m/h during their study.

4. Second ozone addition and GAC filtration

The second application of ozone is the main addition in the plant and is used to facilitate biodegradation of organics in the following GAC filtration step. Agbalika et al. (1985) report an ozone dose varying from 1.4 to 1.6 mg/L and a ten minute contact time. This gave a residual oxidant concentration up to 0.3 mg/L. In their study Agbalika et al.

reported that no enteroviruses were found following this second stage ozonation. They identified this step as the critical barrier ensuring a water free of viral contamination.

There are a total of 12 activated carbon filters, each of the same size as the sand filters. These consist of 6 originally used as sand filters and an additional 6 which were constructed later. The carbon bed depth is 1 m and the grain size is reported as 1 mm. The filtration rate is reported as 9 m/h at a total production rate of 270,000 m^3 per day.

The basic purpose of the carbon filtration step is to allow for biological elimination of ammonia and organic micropollutants. Following a study carried out at the plant, Albat et al. (1986) report that bacterial action in this step is particularly effective for removing organic nitrogen compounds. The physical arrangements are such that all 24 filters could be operated in parallel if an exceptional demand for water were to arise.

5. Post-ozonation

The purpose of the third ozonation step is to provide destruction of viruses, although the study by Agbalika et al. (1985) indicates that this is achieved in the second ozonation step. In this third step, ozone is applied according to standard French practice for virucidal treatment, namely an ozone residual of 0.4 mg/L following 4 minutes contact time.

6. Final Disinfection

The final step of treatment is disinfection for distribution system protection. Figure 10 shows this being accomplished with sodium hypochlorite but as of 1982 it was being effected with chlorine dioxide (Agbalika et al., 1985). These authors report a value of 0.1 mg/L but it is not clear whether this is a residual or a dose.

4.3.5 Effectiveness of Treatment

Table 13 gives the effectiveness of the treatment plant with respect to major water quality parameters. It is evident that good treatment is being obtained.

4.3.6 Treatment Costs

Treatment costs were requested but not provided and efforts will be made to obtain them. Santini (1984) reports that for the Water Authority of the Paris Suburbs as a whole operating costs make up about 60% of total expenditures with the remaining 40% going to investment financing. The selling price of water is set by contract between the Water Authority and the Compagnie Générale des Eaux. The final price is the result of combining a base rate, set at the beginning of the contract, and a variation formula which takes into account changes in economic conditions and operational costs. By means of this formula the price of water is readjusted theoretically twice a year.

4.3.7 Implications for Edmonton

As with Zurich, the direct applicability of process details from Méry-sur-Oise to Edmonton is not great, because of the major differences in raw water quality. However, the general scheme of treatment is relevant.

As with the other two plants, multi-step treatment is provided. Ozone is used as the major oxidant and disinfectant, and another disinfectant is introduced only at the end of the process for distribution system protection. Granular activated carbon is used in combination with ozone, specifically to achieve biological treatment.

Specific treatment features worthy of possible consideration for Edmonton would include raw water storage, although the need for this in Edmonton is not nearly as great as at Mefy. Additional noteworthy process features are the use of water aluminum chloride instead of conventional alum for coagulation

Table 13. Effectiveness of Treatment for Major Water Quality Parameters at the Méry-sur-Oise Plant (Sept. - Dec. 1980).

		Reported Values		
Parameter	Units	Raw Water	After Sand Filtration	Finished Water
Turbidity	not readable	217	1.30	-
Suspended solids	mg/L	45.5	0.050	-
NH_4^+-N	mg/L	1.03	0.010	<0.05
TOC	mg/L	4.60	2.59	2.07
UV-extinction	not given	0.0995	0.0665	0.0207
Coliforms	per 100 mL	17,500	600	0.1

and the provision of separate compartments for the addition of various chemicals in Step 2 of the treatment.

5. CONCLUSIONS

1. Results obtained by the three advanced European drinking water treatment plants reviewed in this report show that it is possible to produce very high quality finished water from both good and impaired sources. In one case (Méry-sur-Oise) the raw water is of considerably lower quality than the North Saskatchewan River.

2. This high quality finished water is achieved by the application of advanced treatment techniques emphasizing physical and biological as opposed to chemical processes. Although the emphasis is different at each of the three plants, all employ a number of treatment steps in series. All three plants (considering the changes planned at Zurich) use ozone as the primary oxidant/disinfectant. There are at least two ozone addition points in the process. Chlorine or chlorine dioxide are used only for distribution system protection and are added only at the end of the treatment process.

3. All three plants use granular activated carbon (GAC) adsorption for organics removal.

4. In contrast to North American practice biological processes are encouraged in the treatment plants for the removal of ammonia and organic compounds. The biological activity is generally achieved by ozone

addition prior to GAC but may also occur in sand filters. In some cases the main purpose of having GAC is to permit this biological activity. In all cases biological removal provides a significant enhancement of the carbon bed life. Because of the TOC reductions obtained, the treated water is relatively stable biologically. This allows low final oxidant doses for distribution system protection.

5. The processes described above are generally based on a philosophy of avoiding disinfectant by-product formation and using a number of treatment steps to reduce the levels of organic group parameters such as TOC and TOX. The approach taken is that this strategy will effectively handle any specific organic contaminants which may be present.

6. The experiences at Langenau show the possibility of producing a water with extremely low turbidity from a source with rapidly changing and occasionally very high suspended solids loads. By emphasizing good flocculation, many other process and quality problems have been solved. Of particular note is the commitment to maintaining finished water quality in the face of all raw water conditions, the desire for process understanding and optimimization and the practice of extensive laboratory and pilot testing prior to the implementation of any process changes.

7. Considerably better treated water quality could be provided in Edmonton, particularly during adverse raw water conditions, by the optimization of existing processes and the addition of new ones. For example, in March 1986, average finished water turbidities for the four plants were in the range 0.26-0.41 NTU, with values exceeding 1 NTU at least once during the

month because of process upsets (Edmonton Water and Sanitation, 1986). Better treatment would of course imply additional costs, but these need not be excessive. Improved treatment is of course not the only option for improving finished water quality, and matters such as source improvement should not be overlooked. Any investigation of treatment improvements should proceed with clear goals in mind, based on policy decisions by the appropriate level of authority.

6. RECOMMENDATIONS FOR PROCESSES WARRANTING STUDY

1. Edmonton Water and Sanitation should conduct investigations aimed at improving the level of treatment provided by the existing processes at the E.L. Smith and Rossdale plants. These investigations should have clearly defined goals, should be thorough, and should consist of both bench and pilot scale studies. They would likely last several years.

An initial item to be addressed would be the optimization of coagulation, flocculation and settling for turbidity and organics removal, using existing physical facilities. The effectiveness of secondary flocculation by the addition of a coagulant directly to the filters should be assessed.

The separation of chemical addition points must also be examined.

An additional process step which could later be considered would be a separate high and variable energy rapid mix step for the solids contact units at E.L. Smith. A compact flocculation type process could be evaluated as a pretreatment for times of high raw water turbidity. Optimization of the softening process to reduce carryover to the filters should also be evaluated. The compact flocculation process should perhaps also be investigated for this application.

2. Ozone should be evaluated as the main oxidant/disinfectant. The investigations should include its addition at two or more points in the process.

3. Granular activated carbon should be investigated for adsorption and biological removal. This must be examined in conjunction with ozone and an indication should be obtained from Alberta Environment prior to the study as to the possibility of ultimately introducing biological processes into the full-scale treatment train.

4. Studies should assess the impact of lower turbidities and organic levels in the finished water on the disinfectant residuals necessary to maintain distribution system protection.

7. ORDER OF MAGNITUDE COST ESTIMATES FOR PILOT SCALE STUDIES

Order of magitude cost estimates for the recommended investigations are provided in Table 14.

The cost of the pilot plant was estimated as follows: the stainless steel/glass/Teflon pilot plant constructed under the writer's supervision at Rossdale cost approximately $70,000 for components and an approximately equal amount to design and build. Assuming that the proposed pilot plant would not need to be constructed of these materials an amount of approximately $100,000 is suggested for a pilot plant incorporating existing Rossdale or E.L. Smith process steps plus GAC and ozone. This includes instrumentation for continuous monitoring of pH, turbidity, headloss, etc.

Table 14 Order of Magnitude Cost Estimates for Pilot Scale Investigations

a. Salary Costs

Classification	Salary ($/month)	Time (months)	Cost ($)
Principal investigator	600 (per diem)	50 (days)	30,000
Process specialist	600 (per diem)	10 (days)	6,000
Project manager	2,500	30	75,000
Chemist	2,500	30	75,000
Pilot plant operator	2,000	24	48,000
Assistant operator	1,600	24	38,400
Stenographer	1,800	2	3,600
Draftsperson	1,800	1	1,800
SUB-TOTAL			277,800

b. Disbursements

Pilot plant (installed)	$100,000
Laboratory & pilot plant supplies ($500/mo)	15,000
Specialized analyses (GC/MS, etc.)	20,000
Communications (telephone, telex, etc.)	2,000
Reproduction	2,000
Shipping	2,000
Travel	10,000
SUB-TOTAL	$151,000

Total estimated cost (no overhead on salaries): approx.	$430,000
Total estimated cost (30% overhead on salaries[a]): approx.	500,000
Total estimated cost (100% overhead on salaries[a]): approx.	670,000

[a] No overhead on principal investigator or process specialist.

Once the pilot plant is in operation the major costs are for labour. A pilot plant requires, depending on program complexity and degree of automation, from 1.5 to 2 full time operators. This provides day-shift staffing 7 days a week and assumes that the plant can be checked by treatment plant operators during the evening and night shifts. The additional project staff required depends on the frequency and number of analyses to be performed. For this estimate one full time chemist has been assumed. One full-time engineer would be required to supervise and direct the study on a day-to-day basis. The engineer and chemist would perform the bench scale tests early in the study. Input from a principal investigator and one or more senior process specialists would also be required.

The costs in Table 14 are for a 2 ½ year program consisting of 6 months for bench scale testing and pilot plant construction and 2 years of pilot scale investigations. Salary costs have been calculated for three levels of overhead: 0, 30 and 100%. Only a small component for analyses has been included. It is assumed that the organization conducting the study will have access to most of the instrumentation required and so the cost of analyses is included in the chemist's time.

An order of magnitude cost estimate for the recommended pilot scale studies ranges from $430,000 to $670,000, depending on the level of overhead.

8. ACKNOWLEDGEMENTS

The writer would like to thank the following for providing information:

Dr. M. Schalekamp, director, Zurich Water Supply, Switzerland.

Prof. Dr. H. Sontheimer, Karlsruhe University, West Germany

Prof. Dr. H. Bernhardt, Siegburg, West Germany

Dr. P. Wölfel, Landeswasserversorgung, Langenau, West Germany

Dr. B. Langlais, Trailigaz, Paris, France.

9. REFERENCES

Agbalika, F., P. Hartemann, J.C. Joret, A. Hassen and M.M. Bourbigot (1985). Study of indigenous virus removal at different stages in a drinking water plant treating river water. Water Science and Technology, 17, pp. 211-218.

Albat, M., B. Baleux, B. Picot, J.M. Philipot and J. Bontoux (1986). Evolution de l'azote Kjeldahl et de la biomasse bactérienne le long d'une filière de traitement d'eau potable. Water Research, 20, pp. 237-245.

Bernhardt, H. (1985). Allgemeine Gesichtspunkte bei der Verfahrenswahl für Flockung und Flockenabtrennung. DVGW-Schriftenreihe Wasser Nr. 42, pp. 321-364.

Bernhardt, H. (1986). Personal communication.

Billen G., C. Cavelier, S. Dessery, M. Meybeck and M. Somville (1984). Variations of the River Oise quality when stored in the Méry-sur-Oise basin. Verh. Internat. Verein. Limnol., 22, pp. 1510-1515. (Abstract only reviewed)

Cavelier, C., M. Caillibotte, F. Damez and O. Pascal (undated). Optimization of the design and operation of a storage basin at the Méry-sur-Oise water treatment plant. Presented at: "Qualité de l'eau dans les retenues", Journée de l'Association Pharmaceutique Francaise pour l'Hydrologie, March 1st, Paris, France (Abstract only reviewed).

Edmonton Water and Sanitation (1986). Water Treatment and Quality Control, Monthly Report for March.

Landeswasserversorgung (undated #1). Trinkwasser von der Landeswasserversorgung (Stuttgart).

Landeswasserversorgung (undated #2). Wie gut ist unser Trinkwasser? (Stuttgart).

Méry-sur-Oise Treatment Plant (1981, 1983). Process decumentation provided to technical visitors.

Santini, A. (1984). Water supply in the greater Paris area: Problems and solutions. Presented at International Symposium Metropolis, Paris, France, Oct. 10-12.

Schalekamp, M. (1986). Reduction of the hygienically doubtful chlorine compounds by pre- and intermediate oxidation with ozone, chlorine and chlorine dioxide. Presented at Second National Conference on Drinking Water, Edmonton, Canada, April 7-8.

Weindel, W. and H. Sontheimer (1971). Flockungshilfsmittel in der Wasseraufbereitung. GWF 112, pp. 76-80.

Werner, G. (1969). Erfahrungen bei der Aufbereitung von Donauwasser durch Flockung und Filtration. Jb. Vom Wasser 36, pp. 186-215.

Werner, G. (1983). Personal communication and notes for technical visitors.

Wölfel, P. (1985). Flockung und Sedimentation (mit und ohne Schlammkontakt). DVGW-Schriftenreihe Wasser Nr. 42, pp. 173-188.

Wölfel, P., G. Werner, and D. Flinspach (1982). Die neue Kompaktflockungsanlage des Zweckverbandes Landeswasserversorgung Stuttgart - Entwicklung, Bau, Einsatzmöglichkeiten und Betriebserfahrungen. DVGW-Schriftenreihe Wasser Nr. 104, pp. 67-91.

Wurster, E. and G. Werner (1971). Die Leipheimer Versuche zur Aufbereitung von Donauwasser. gwf-wasser/abwasser 112 H. 2, pp. 81-90 and H.4, pp 193-199.

APPENDIX 1

Terms of Reference

Sub-contractor: Dr. P.M. Huck,
Professor of Civil Engineering
University of Alberta

Tasks:
2.1.1 Physical, Chemical & Microbiological Quality,
4.4 European Treatment Practices,
5.3.3 Finished Water Quality

Task Descriptions:

2.1.1 Physical, Chemical & Microbiological Quality

1. Assemble via personal contact and literature sources, data on the physical and chemical water quality of raw water sources from 3 or more locations in Europe which may be logically compared to the North Saskatchewan River.
2. Tabulate and summarize data in a format, to be provided, to allow comparison with data obtained for the North Saskatchewan River upstream of the Edmonton water intakes.

4.4 European Treatment Practice

1. Visit the Edmonton Rossdale and E.L. Smith water treatment plants and review, in detail, the processes and practices employed.
2. Constructively critique practices and processes observed with recommendations for specific practices and procedures to achieve improvements.
3. Review advanced water treatment practice at 3 or more European locations comparable in size to Edmonton. Facilities which have overcome chronic taste and odour problems would be the highest priority in site selection.

4. Document the rationale, in terms of relevance to Edmonton, for the facilities selected for study.
5. Describe the processes used and the range of performance obtained.
6. Describe operating procedures used which are critical to optimum performance
7. Relate the facilities and procedures described to application in Edmonton with descriptions of any major limitations which may be encountered.
8. Document treatment costs for each facility and relate these to application of similar processes in Edmonton.
9. Provide recommendations for any processes which warrant study for application in Edmonton along with order of magnitude estimates for pilot study resources.

5.3.3 Finished Water Quality

1. Assemble via personal contact and literature sources, data on the physical, chemical and microbiological water quality of finished water from 3 or more locations in Europe which may be logically compared to Edmonton.

2. Summarize any standards and water quality criteria which these facilities must meet along with an indication of the frequency and severity of excursions.

3. Tabulate and summarize data in a format, to be provided, to allow comparison with data obtained for Edmonton finished water quality.

APPENDIX 2

Sample Request for Information

P.M. Huck, Ph.D., P.Eng.
11720 - 37B Avenue
Edmonton, Alberta, Canada
T6J 0K4
Telephone: (Bus.) (403) 432-4738
Telex: 0372979

January 31, 1986

Dr. M. Schalekamp
Director, Zurich Water Supply
Hardhof 9
Postfach
CH 8023
Zurich, Switzerland

Dear Dr. Schalekamp:

The City of Edmonton and the Province of Alberta have recently retained an independent consultant to examine the quality of the City's treated drinking water. The rationale for the study is outlined in the attached "Statement of Need" but basically stems from an annual hydrocarbon-related taste and odour episode in the spring coupled with occasional reports of elevated concentrations of specific organics in the water. One of the two treatment plants is located in the middle of the city and is subjected to upstream discharges such as accidental spills, storm water discharges and in particular spring runoff.

I have been retained as a specialist to address one aspect of this investigation: the comparison of Edmonton's treatment practices to those in advanced Western European plants and a comparison of raw and finished water quality for those facilities to that for Edmonton. My terms of reference are attached. Although there is no budget for me to visit European plants, I have previously visited a number of plants in France and West Germany.

I wish to include one example of Swiss practice in this comparison and based on information available to me I have selected the Zurich water supply. My purpose in writing is to enquire whether you could arrange to provide me with the necessary documentation and information required to complete the following parts of my task:

Task 2.1.1 - Item 1
Task 4.4 - Items 5,6,7,8
Task 5.3.3 - Items 1,2.

While I recognize that your supply is composed of both lake and groundwater sources I would propose to concentrate on the treatment of the lake supplies. While this would not be directly comparable to a river supply such as Edmonton's my understanding is that improvements to your treatment practice were initially motivated by a hydrocarbon-related taste and odour incident in 1967. No doubt your current treatment is designed to provide a measure of protection against future recurrences of such incidents.

I feel that you would be the best judge of what information you have available which would assist me and therefore I will not ask for specific documents. A list of the analyses performed for Edmonton is included to give you an indication of the type of information on raw and treated water quality I would require. I have the 1983 annual report of the Zurich water supply in my files and I am aware of your various published papers on the development of new treatment processes although I do not have copies of these. I would ask your permission to use your submitted presentation to our April conference as an unpublished working document.

I read both German and French well so you may send me anything in those languages which is not available in English.

I have a budget of up to $200 Canadian which can be used to cover your costs of photocopying, shipping and if necessary charges for one of your staff member's time to assemble the appropriate information. Please invoice me for any such costs you may incur. Although my private address appears at the top of this letter, for convenience you may send the information to me care of:

Department of Civil Engineering
University of Alberta
Edmonton, Alberta, Canada
T6G 2G7

Let me thank you in advance, Dr. Schalekamp, for your assistance in this matter. I look forward to the chance to discuss these matters further with you in April if that is required.

Yours very truly,

P.M. Huck, Ph.D., P.Eng.

PMH/ds
Encl.

APPENDIX 3

Detailed Water Quality Data

Table A3-1. Raw and Finished Water Quality Lengg Plant, Zurich, Switzerland (1985 data)

		Measured Values					
		Raw Water			Finished Water		
Parameter	Units	Avg.	Min.	Max.	Avg.	Min.	Max.
a. Overall Parameters							
Temperature	°C	5.8	4.3	7.1	5.8	3.6	7.8
Turbidity (Extinction)	420 nm d=1 m	.31	.22	.44	.06	.02	.08
Turbidity[a] (Scattering -90°)	FTU	.28	.17	.50	-	-	-
Particles < 3 µm[a]	per mL	51,505	30,584	124,421	-	-	-
Particles 3-10 µm[a]	per mL	8,556	3,681	16,942	-	-	-
Particles >10 µm[a]	per mL	1,255	590	2,163	-	-	-
Oxidants[a]	mg/L	0.56[b]	0.37[b]	0.76[b]	0.04[c]	0.02[c]	0.07[c]
Redox potential[a]	mV	740	670	795	765	720	780
pH		7.85	7.62	7.99	8.06	7.82	8.96
Equilibrium pH[a]		8.15	8.12	8.19	8.11	8.07	8.16
Free CO_2	mg/L	3.8	1.8	4.8	2.1	0.9	3.1
Conductivity	µS/cm	260	245	280	265	255	280
Carbonate hardness[d]	°fr	12.3	12.1	12.6	12.8	12.5	13.0
Calcium hardness[d]	°fr	12.2	12.0	12.4	12.8	12.4	13.1
Magnesium hardness[d]	°fr	1.5	1.0	1.9	1.7	1.4	2.0
Total hardness[d]	°fr	13.7	13.2	14.1	14.5	14.1	14.9
Silicic acid[a] as S_iO_2	mg/L	2.2	1.5	2.6	2.0	1.6	2.4
Chloride	mg/L	4.3	4.1	4.4	5.3	5.2	5.4

Table A3-1 continued

		Measured Values					
		Raw Water			Finished Water		
Parameter	Units	Avg.	Min.	Max.	Avg.	Min.	Max.
a. Overall Parameters (continued)							
Dissolved oxygen	mg/L	8.9	6.5	10.6	11.5	10.3	13.1
D.O. saturation	%	74	56	84	98	94	102
Ammonia nitrogen[a]	µg/L	<5	<5	<5	<5	<5	<5
Nitrate nitrogen	µg/L	820	785	855	850	820	870
Nitrite nitrogen[a]	µg/L	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Phosphate phosphorus[a]	µg/L	53	47	64	44	33	55
b. Metals and Metalloids							
Aluminum	µg/L	40	5	70	170	105	300
Arsenic	µg/L	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Lead	µg/L	0.5	0.3	0.9	0.2	<0.1	0.3
Cadmium	µg/L	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Chromium	µg/L	<1	<1	1	<1	<1	<1
Iron	µg/L	20	10	35	5	<5	10
Copper	µg/L	11	4	18	5	2	14
Manganese	µg/L	3	3	4	<1	<1	<1
Molybdenum	µg/L	4	2	7	2	<1	4
Mercury	µg/L	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Zinc	µg/L	25	15	35	5	<5	10
c. Organic Parameters							
UV Extinction	254 nm/ 1m	3.0	2.8	3.2	1.0	0.7	1.2

Table A3-1 continued

Parameter	Units	Measured Values					
		Raw Water			Finished Water		
		Avg.	Min.	Max.	Avg.	Min.	Max.
c. Organic Parameters (continued)							
Oxidizability $KMnO_4$	mgO_2/L	1.1	0.9	1.2	0.6	0.5	0.7
DOC	mg/L	1.20	1.05	1.30	0.85	0.70	1.00
Chloroform[a]	µg/L	4.4	1.2	7.3	4.3	1.2	6.1
Bromodichloromethane[a]	µg/L	0.7	ND	1.8	0.9	ND	1.4
Dibromochloromethane[a]	µg/L	0.1	ND	0.2	0.1	ND	0.4
Total THMs[a]	µg/L	5.1	1.8	9.0	5.2	1.6	7.9
Carbon tetrachloride[a]	µg/L	ND	ND	ND	ND	ND	ND
Perchloroethylene[a] (C_2Cl_4)	µg/L	ND	ND	ND	ND	ND	ND
Trichloroethylene[a] (C_2Cl_3H)	µg/L	0.3	ND	2.8	0.1	ND	1.6
Fluoroanthene[e] (Fluoranthen)	ng/L	11.6	6.0	27.5	11.7	4.3	36.4
3,4-Benzofluoroanthene[e] (3,4-Benzfluoranthen)	ng/L	2.1	0.7	5.7	1.9	ND	7.4
3,4-Benzopyrene	ng/L	1.4	0.2	2.6	1.1	ND	6.8
1,12-Benzopyrelene[e] (1,12-Benzperylen)	ng/L	7.2	0.6	21.4	5.7	0.8	28.8
11,12-Benzofluoroanthene[e] (11,12-Benzfluoranthen)	ng/L	0.5	ND	2.1	0.2	ND	0.7
Indenopyrene[e] (Indenopyren)	ng/L	1.1	ND	2.4	0.9	ND	6.3
Total PAH	ng/L	24.0	8.1	46.8	21.5	6.9	67.2

Table A3-1 continued

		Measured Values					
		Raw Water			Finished Water		
Parameter	Units	Avg.	Min.	Max.	Avg.	Min.	Max.
d. Biological Parameters							
Phytoplankton[a] (>20 μm)	per mL	174	40	527	-	-	-
Phytoplankton[a] (2-20 μm)	per mL	1,636	274	5,912	-	-	-
Phytoplankton[a] (<2 μm)	per mL	1,743	260	7,600	-	-	-
Phytoplankton[a] biomass	mg/L	0.551	0.137	1.325	-	-	-
Nematodes[a]	per m^3	0	0	0	-	-	-
Phyllopoda[a]	per m^3	958	15	4,314	-	-	-
Total Copepoda[a]	per m^3	1,107	7	2,679	-	-	-
Nauplii[a]	per m^3	910	2	2,320	-	-	-
Canthocamptus[a]	per m^3	0	0	0	-	-	-
Total animals[a]	per m^3	2,975	23	5,560	-	-	-
Total bacteria	per mL	102	10	400	2	0	120
Total Coliforms	per 100 mL	4	0	40	0	0	0

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[a] Raw water values measured after chlorination

[b] Calculated as Cl_2

[c] Calculated as ClO_2

[d] It is assumed that °fr stands for French degree of hardness.
1 French degree of hardness = 10 mg/L $CaCO_3$

[e] Translation could not be confirmed. Original German given in brackets.

ND - not detectable (no detection limits given)
- - not measured

Note: Data for the Moos plant are very similar and are not included.

Source: Zurich Water Supply

Table A3-2. Raw and Finished Water Quality - Langenau

		Raw Water (1985)			Finished Water[a]
Parameter	Units	Average	Minimum	Maximum	Average
Temperature	°C	10.4	5.7	16.3	9.6
Odour		not reported			ND
Colour (Pt)	mg/L	15.5	9.6	28.8	0.2
Turbidity	FTU	8.0	1.0	54	0.04
Conductivity	µS/cm (20°C)	470	360	560	510
Ionic strength	$\mu S \times 10^{-3}$	8.7	6.5	9.7	-
pH (electrode)		7.50	7.21	7.72	7.33
Free CO_2	mg/L	18	10	35	29
Total hardness	degrees[b]	15.0	11.15	16.8	18.0
Carbonate hardness	degrees[b]	12.9	9.7	14.6	14.9
Non-carbonate hardness	degrees[b]	2.1	1.3	2.75	-
Total iron (Fe^{2+}/Fe^{3+})	mg/L	0.10	0.059	0.209	0.006
Manganese	mg/L	0.03	0.0021	0.130	0.001
Calcium	mg/L	82	59	93	111
Magnesium	mg/L	15.2	12.2	18.0	11.0
Sodium	mg/L	9.6	5.9	13.4	4.3
Potassium	mg/L	2.1	1.3	3.3	1.3
Sulfate	mg/L	28	14	34	26
Chloride	mg/L	23	16	30	21
Nitrate (NO_3)	mg/L	9.7	3.7	12.7	21
Nitrite (NO_2)	mg/L	0.09	0.05	0.16	ND
Ammonia (NH_4^+)	mg/L	0.20	0.05	0.45	ND
Orthophosphate (PO_4^{3-})	µg/L	360	77	543	91[c]
Polyphosphate (PO_4^{3-})	µg/L	31	11	102	-
Dissolved Oxygen	mg/L	6.6	3.7	9.8	10.1
Oxygen saturation	%	60	37	82	-
DOC	mg/L	2.7	1.8	3.2	0.7
UV extinction (254 nm)	m^{-1}	6.9	4.6	9.3	1.1
$KMnO_4$ demand (after filtration)	mg/L	12.2	7.9	13.6	2.1

Table A3-2 continued

Parameter	Units	Raw Water (1985)			Finished Water[a]
		Average	Minimum	Maximum	Average
Oxygen demand (O_2)	mg/L	3.1	2.0	3.4	-
Cyanide (CN^-)	mg/L	0	0	0	<0.001
Free chlorine	mg/L	-	-	-	0.015

[a] Consisting of part groundwater. Values for Rotenberg reservoir in Stuttgart. Exact year not given, but believed to be 1980.

[b] One German degree of hardness = 17.86 mg $CaCO_3$/L

[c] Total phosphorus

ND Not detectable (detection limits not given)

Source: Zweckverband Landeswasserversorgung

Table A3-3 Additional Finished Water Data for Langenau[a]

Parameter	Units	Reported Value	Percentage of Allowable Limit
Arsenic	µg/L	0.15	0.4
Lead	µg/L	<1	<2.5
Cadmium	µg/L	0.02	0.3
Chromium	µg/L	0.4	0.8
Cyanide	µg/L	<1	<2.0
Fluoride	mg/L	0.08	5.3
Nitrate	mg/L	21	23.0
Mercury	µg/L	<0.01	<0.25
Selenium	µg/L	<0.05	<0.6
Zinc	µg/L	10	0.5
PAH	µg/L	<0.01	<4.0

[a] believed to be for 1980

Source: Landeswasserversorgung (undated #2)

Table A3-4. Raw and Finished Water Date for Méry-sur-Oise (Sept. - Dec. 1980)

Parameter	Units	Average Reported Value Raw	 Finished
Turbidity	not readable	217	-
SS	mg/L	45.5	-
Dissolved oxygen	mg/L	8.72	9.67
pH		8.09	7.53
Ammonia nitrogen	mg/L	1.03	<0.05
Kjeldahl nitrogen	mg/L	0.736	0.405
$KMnO_4$ demand	mg/L	3.87	0.69
UV extinction	not given	0.0995	0.0207
TOC	mg/L	4.60	2.07
E.Coli	per 100 mL	2,455	0
Total coliforms	per 100 mL	17,447	0.10
Clostridium	per 100 mL	1,118	0.035
Streptococcus	per 100 mL	1,424	0
Total plate count[a] (37°, 24h)	per mL	6,735	7.65
(20°, 72h)	per mL	69,077	31

[a] Presumed translation of "germes banales", literally, "ordinary bacteria"

APPENDIX 4

Reports of Edmonton Site Visits

To: S.E. Hrudey December 18, 1985

From: P.M. Huck

Re: Rossdale and E.L. Smith site visits - December 16, 1985

1. My scope of work will not permit me to submit a separate detailed report on these site visits. However I feel it would be useful for me to review and comment on Paul Foley's report, provided he agrees. The following points summarize my specific comments following the visits.

2. Addition of chemicals. In both plants a number of chemicals (i.e. PAC, CLO_2, ammonia and alum) are added virtually simultaneously at the beginning of the process. While this practice has been necessitated by plant constraints in the past, it should be reviewed, with a view to process optimization. For example, it is possible that the PAC is being blinded at least partly by the alum. In addition it is known that activated carbon can catalyze reactions between chlorine dioxide and organics. Since chlorine dioxide reportedly does not react with ammonia, the chlorine smell detected in the first compartment flocculator at Rossdale may be due to inefficient chlorine dioxide generation.

3. PAC operating procedure. This should be critically reviewed to ensure its adequacy (I am requesting their detailed procedure). The reasons for the establishment of a 5 ppb response level should be reviewed. What action is taken if the maximum carbon dose which can be fed is inadequate to deal with the particular situation?

I have two particular concerns with this method of response to organics spills. In the case of the Rossdale plant, it is not clear whether inadequately treated water can be diverted before it reaches the distribution system (I will ascertain this). In the case of the E.L. Smith plant with no GC capability on site, only compounds which lead to a noticeable odour will be detected. Even at the Rossdale plant, if a spill occurs btween GC runs, only an odour will provide a means of detection.

4. Turbidity on top of filters at Rossdale. I would like to see an effort made to improve the turbidity reductions prior to filtration. This would involve an optimization of the softening step which I do not believe has been systematically done. While the current practice may provide satisfactory operation, better plant performance could be achieved by attention to this matter.

5. Possible disruptions in filter beds at E.L. Smith. The bubbling observed in certain sections of the filter during backwashing on December 16th is indication of possible disruption in the bed or underdrain system. I observed similar behaviour in one of the older filters at E.L. Smith in July of 1983; my recollection is that it was worse.

6. Optimization of processes with respect to organics removal. Based on Karim Kassam's statements and my own understanding I do not believe that the overall treatment process has ever been properly optimized for the removal of either background organics or synthetic compounds which may be present on a transient basis.

7. Process development capabilities. Many of the above comments lead me to the conclusion that what is required is someone with senior responsibilities and an appropriate technical background to be responsible for process development at the Edmonton plants. Such a person would require a combination of good academic grounding, perhaps in Chemical Engineering but certainly in Environmental Engineering unit operations, combined with considerable practical experience. While I cannot suggest how such an individual should be established within the organizational structure, he/she would have to have sufficient authority and perhaps operating responsibility to see that the results of his/her work were placed into practice.

I will be happy to investigate any of these matters in further detail, if you feel it is appropriate.

Peter

PMH/ds

P.M. Huck

To: S.E. Hrudey February 3, 1986

From: P.M. Huck

Re: Activities in January

Slightly more than half the time was spent on the site visit and discussions with Vern Snoeyink. Notes from discussions with Vern, Doug Murdoch, Myron Laholla and myself on January 13 follow:

1. Taste and odour operating procedure (some aspects)
    - since last year they have maintained >0.15 mg/L ClO_2 residual (higher than before).
    - they can "handle" (at Rossdale) up to about 80 µg/L hydrocarbons (volatiles by their GC method). With PAC (and ClO_2) they can get down to 8-10 µg/L in the finished water, a level which apparently does not produce taste and odour complaints.
    - maximum carbon feed is currently 20 mg/L at Rossdale, although it will be 25 mg/L soon. At this rate they see the effect on the filters in about 6 hr (runs are reduced from 36 to 8-10 hrs).

2. For E.L. Smith, they rely primarily on UV and colour measurements. The Smith runoff tends to produce a musty odour. Smith is not a good predictor of Rossdale taste and odour since hydrocarbons tend to dominate

at Rossdale. According to Myron, the runoff from March to May produces colour at Smith (from riverbanks, etc.). Mountain runoff produces silt in July.

3. For Rossdale 1 & 2 plants, water must go to the distribution system once it enters the plant. Rossdale #3 and Smith output can be diverted to waste.

4. Myron advises that they are advised of spills in the city as they occur.

5. Myron and Doug reported that the distribution system pipes are very clean, that all turberculation is external. There is reportedly about 1/8" of scale on the inside of the pipes, covered by about 1/4" - 1/8" of soft sludge. Speculation in the discussion was that this was probably an alum deposit or the result of biological activity.

PMH/ds

P.M. Huck

Report on Evaluation of City of Edmonton Water Laboratory

B. Kratochvil

June 1986

Introduction

The City of Edmonton water laboratory performs a large number of daily physical and inorganic tests and analyses for the purpose of monitoring and maintaining drinking water quality. A summary of the tests for one arbitrarily selected month (January 1986) shows (Table 2.2.1a of the monthly report) that from 130 to 390 tests were performed on each of 12 parameters on raw and treated water at each water plant. Typically values are obtained daily on the raw water at the E.L. Smith and Rossdale water treatment plants, and on the treated water at Rossdale No. 1, 2, and 3 plants and at E.L. Smith #4 plant, for 7 parameters (total alkalinity, calcium, color, fluoride, total hardness, pH, and turbidity), and on the treated water for 5 parameters (phenolphthalein alkalinity, ClO_2 residual, ClO_2 yield, free Cl_2, and total Cl_2). In addition a number of tests are performed less frequently than daily (Table 2.2.1b of the monthly report), from twice a week or so to once a month. These include ammonia, chloride, conductivity, sulfate, sulfide, total dissolved solids and total organic carbon. Several more are sent out for external analyses such as radioactivity measurements for cesium-137, iodine-131, radium-226, strontium-90, and tritium, ammonia (as N), cyanide, nitrite, and nitrate. This list does not include values calculated from data obtained in the other tests such as agressiveness index and magnesium as $CaCO_3$, automatically recorded temperature values, odor tests conducted by operators, or organic measurements (THM's, volatile organics, and UV). Additionally, 16 reservoirs in the distribution system were tested for 12 physical and inorganic parameters during the month, and 14 for ClO_2 and residual chlorine one to three times during the month.

Some of the simpler tests were performed by plant operators; the remainder were performed by laboratory staff. The samples from the reservoirs were collected by a laboratory assistant assigned the job of sample collector. The sample containers are prepared in the analytical laboratory.

Assessment of Monitoring Practice

The test procedures used by the City of Edmonton Water Laboratory for the parameters listed above were evaluated by detailed study of the laboratory methods manual, and by several visits to the laboratory, including discussions of the methods used with laboratory personnel. This evaluation is divided into 3 sections: measurements of chlorine dioxide and related chlorine species, measurements of the physical and inorganic parameters, and measurements of some organic parameters. The chlorine species tests are considered separately because they comprised a special problem area that was handled essentially independently by the laboratory staff. The organic analyses, although not included in the original task assignment, were evaluated to some extent in conjunction with the other tests, and so evaluation of some aspects of this area, based on limited study, are included here.

In general the procedures used by the laboratory are those accepted as standard test procedures in the US and Canada. Almost all are based on methods described in standard works, such as Standard Methods for the Examination of Water and Waste Water (American Public Health Association, American Water Works Association, Water Pollution Control Association, 14th Edition, 1976 or 15th Edition, 1980); Annual Book of ASTM Standards, Part 31, Water, American Society for Testing and Materials; Methods for Chemical Analysis of Water and Wastes, U.S. Environmental Protection Agency; or Methods Manual for Chemical Analysis of Water and Wastes, Alberta Environment Centre). A procedures manual was just being written during the time this evaluation was performed, in essence a first draft was reviewed. Individual sections of the manual had been assigned to various staff members, a reasonable procedure, but one which produced considerable unevenness in treatment. The writeups included precision and accuracy information for a set of measurements on as-received and spiked samples. This is a commendable inclusion. The major item missing from all procedures was how and where to log the results of the tests. This should be included in a revised version.

Some of the procedures appeared to be copied verbatim from the standard methods manuals and did not include needed details on how the procedure is adapted to the Edmonton laboratory. Others omitted needed information, such as drying times and temperatures. Questions and corrections were annotated throughout the manual. An edited version is now being prepared.

The manual includes, in addition to a procedures section, sections on housekeeping, safety, sampling, and quality assurance.

Evaluation of Current Laboratory Performance

The laboratory has had to respond to increasing demands for analytical data as new questions arise concerning levels of trace contaminants recently proposed to constitute health hazards. Some of the concentration levels of concern are far below what was even detectable with state-of-the-art analytical methods a few years ago. Under these conditions the laboratory has provided the quantity of analyses requested of it, but has not been able to provide the quality needed.

In some instances two methods are used for a parameter; for example, fluoride is measured by an on-line monitor, and is also determined manually because the on-line system is not trusted. Such duplication should be avoided by trouble shooting or revising the on-line system so that it provides reliable data that does not require constant duplication of work. Manual analyses should be required only for periodic checks of the automatic system.

The precision and accuracy of the measurements could only be evaluated indirectly. Accuracy must be determined by comparison of analytical results with known values of standards or reference samples; the laboratory does not run standards or test samples obtained externally to assess their performance. Single data sets provided by the laboratory on recovery of spikes from water samples indicate that the accuracy of the measurements is in general acceptable, but this was done only once, and the analyst knew beforehand the size of the spike. The most reliable evaluation of laboratory performance is obtained by the introduction of blind external test samples into a set of routine analyses such that they are not accorded special treatment. This is difficult to do in a laboratory where samples are often collected by the analyst from a tap in the laboratory. For those tests routinely performed by the plant operators the laboratory has instituted a policy of introducing weekly a sample prepared by the laboratory supervisor for which the operator must submit a value. This is a valuable quality control procedure and its continuation is recommended not only for operators but also for all other tests reported insofar as is feasible. This includes those tests which are sent to outside laboratories such as those for radiochemical species. More standards and blind test samples should be run. These can be obtained from an external agency such as the EPA through their round-robin laboratory assessment program, from Alberta Environment, or prepared by other reliable outside organizations.

Chlorine Dioxide Analyses

The determination of levels of chlorine dioxide and other chlorine species in water undergoing treatment has been a significant problem for the water laboratory from the time use of ClO_2 was instituted in the process in 1982. Initially ClO_2 was measured in the process streams spectrophotometrically at 360 nm. The results were apparently in error for some time owing to incorrect calibration curves. For the low concentration range of ClO_2 in the water leaving the plants, an amperometric titration with phenylarsine oxide at pH 7 was employed, but precision was poor.

Literature Review. Methods for the determination of ClO_2, ClO_2^-, ClO_3^-, and Cl_2, at low levels in water are available, but little has been published about the effects of chloramines on the analytical results. The problem has been brought under partial control in the Edmonton system since about the end of 1985, but an accurate analysis of low levels of chlorite on a routine basis is still not available.

Close monitoring of the level of ClO_2 is important because the primary product upon reaction of ClO_2 with oxidizable substances such as organic compounds is ClO_2^-, for which the health effects are not well understood. Of the methods for the determination of ClO_2, ClO_2^-, and Cl_2, the one in Standard Methods for the Examination of Water and Wastewater, 14th Edition, 1976, has been shown to be inaccurate (see Aieta, Roberts, and Hernandez, Jour. AWWA, January 1984, 64-70 and references therein). Aieta et al. suggest modifications to the standard methods procedure that seem to give satisfactory results, but the method is slow and requires careful technique by the analyst. Gordon and coworkers (see Jour. AWWA, July 1984, 98-101, and references therein) propose modified iodometric methods for chlorite, hypochlorite, chloride, and chlorate, using potentiometric titration by thiosulfate of the iodine produced after various treatments as the measurement process. The US EPA has prepared both titrimetric and colorimetric procedures for the measurement of ClO_2, free and combined available residual chlorine, and chlorite by reaction with N,N-diethyl-p-phenylenediamine (DPD) to produce a red color which is titrated with ferrous ammonium sulfate to colorless in the titration procedure, or read directly in a spectrophotometer in the colorimetric procedure. A comparison of the DPD method with the iodide titration method for mono- and di-chloramines by the EPA-Cincinnati laboratory (see data sheet), shows that the DPD method agrees with the iodide method for total chloramines, but the DPD procedure allows the mono- and dichloramines to be estimated individually in a mixture. A spectrophotometric method for ClO_2 based on the oxidation of chlorophenol red (Harp, Klein, and Schoonover, Jour. AWWA, October 1982, 387-388) is claimed to be free from interference by hypochlorite, chlorite, chlorate, or chloramines. A voltammetric membrane electrode also has been proposed for the measurement of low concentrations of ClO_2 without interference from hypochlorite, chlorite, or chlorate (Smart and Freese, Jour. AWWA, October 1982, 530-531). Such a system could be well used for continuous automated monitoring.

Present Practice in the Edmonton Water Laboratory. The laboratory has developed a potentiometric titration method using commercial titrators and phenylarsine oxide as titrant that is more rapid than the Standard Methods or Aieta et al. procedures, and which gives adequate precision. This "two step" method appears to be in a satisfactory state of statistical control at the present time, and is used by the plant operators to measure total oxidant. The method still requires additional study, however, to establish its accuracy and ruggedness under plant conditions.

Organic Analyses

The organic analyses by purge-and-trap and by head-space gas chromatographic (GC) methods, and the ultraviolet (UV) spectrophotometric organic measurements, were evaluated. The identification of organic compounds in the GC analyses is based on comparison of retention times with those of known compounds.

Because many compounds may have the same retention time, identification should be confirmed by another method such as mass spectroscopy (MS). The UV absorbance measurements are used as an indicator of the amount of organic material in the raw water; absorbances have been shown by the laboratory to correlate with GC total peak area measurements. Thus UV measurements provide a rapid and simple indicator of organic contamination. The method provides no information on which compounds are present, however, its sensitivity for different compounds depends on the wavelength used, and the procedure is sensitive to bias from adsorption of UV absorbing compounds on cell walls. Therefore, although it may be useful as a crude monitor or warning device upstream, it does not provide specific information.

In summary, as presently used the GC and UV measurements give a qualitative indication of the general level of organic substances present in the water, but do not provide either identification or accurate quantitation. Because the GC methods used are aimed at the more volatile organic components in the water, consideration should be given to the introduction of extraction methods to identify and measure less volatile substances.

Laboratory efforts to obtain more complete identification and quantitation of the compounds present in problem waters should be increased. Identification of major GC peaks by MS, and quantitation as needed by running standards, is recommended. Extraction procedures to test for less volatile organic compounds should be introduced. The use of a flow-through UV instrument as an upstream continuous monitor to give early warning of increases in the level of organic contaminants in the water supply may be worth considering.

Laboratory Analytical Capability

The measurement instrumentation and equipment available in the laboratory is in general adequate. Some of the instruments, such as colorimeters, turbidimeters, etc., appear to have seen much use, but are well maintained and are functioning adequately. In several areas instrumentation is excellent and essentially at the state-of-the-art. Examples of instruments in this class are the two gas chromatographs, ion chromatograph, automatic titrator, and UV-visible spectrophotometer. Present laboratory space is crowded, creating congestion and possible safety hazards. These problems should be less severe when the lab space changes presently underway are completed. The separation of the present laboratory office and work areas is inconvenient and inefficient for some of the staff.

An area in which the lab requires major upgrading is in data collection and processing. High priority should be given to installing a laboratory information management system. A computer should be acquired with software for data recording and report production. The system should include the capability of being interfaced with all the laboratory instruments, and ultimately with on-line monitoring instrumentation in the plants. A system of this kind will reduce errors in instrument reading and data recording, as well as in data transfer from log books to reports. It also will result in savings in time and manpower in the long run. Although only a fraction of the present instruments could be directly interfaced with a computer, others can be adapted for data transfer, and replacement instrumentation can be purchased with built-in provision for interfacing. (Most new instruments contain RS-232 interfaces for standardized information transfer.) In many cases output from the instruments can be sampled by the computer on command, and in some instances instrument operation can be controlled by the computer. Although initial costs will likely be relatively high, improved data quality and long term savings in manpower make the introduction of such a system highly desirable.

The question of whether the laboratory should obtain a GC-MS has been raised, since this instrument would allow identification of trace organic compounds in both raw and treated water. Data in this area is critical to identifying and monitoring compounds that cause taste and odor problems, as well as other compounds of possible hazard to health. Purchase of such an instrument at this time is not recommended, however, because of the major commitment required in instrument costs and in expert manpower before useful information can be obtained. Developments in newer, less costly, MS-based GC detectors such as the Finnigan ion trap are proceeding rapidly, making it likely that cost will drop rapidly while reliability and ease of operation will improve. This rapidly evolving area should be monitored closely; some time in the next few years a GC-MS instrument suitable for routine monitoring will likely become available, and at that time the possibility of purchase should be reassessed. For the time being the required data should be obtained by contracting out analyses to Alberta Environment or to commercial laboratories.

Autosamplers, flow-through monitors, and similar semi-automated instrumentation should be used wherever feasible to improve efficiency.

Quality Assurance in the Laboratory

The goal of an analytical laboratory is timely production of accurate data of the required precision. To ensure this a detailed quality assurance program must be in place. The Edmonton Water Laboratory has a start on quality assurance but it is well behind current good laboratory practice. Some quality control checks are run on measurements made by the plant operators, instruments are calibrated intermittently, and some reference samples are run to check procedures, but planning is haphazard. Quality assurance operations should be greatly expanded and placed on an organized, systematic basis. The laboratory methods manual now in preparation should include, in addition to calibration procedures and preparation of calibration curves, information on the frequency with which these operations must be done, direction on the running of duplicates and standards, and report formats. More use should be made of blind test samples to ensure that instruments, procedures, and worker performance are all satisfactory. Participation in interlaboratory round-robin testing of EPA, Alberta Environment, or other samples should be required to obtain an objective measure of laboratory performance. All quality assurance operations should be made the responsibility of a single individual.

More interaction with the staff of other water utility laboratories who have similar water supplies, such as those at Calgary or Regina, should be fostered with the aim of sharing information on possible solutions to analytical problems.

Staffing and Manpower in the Water Laboratory

Background. The water laboratory as of February 1986 had a staff of 10 permanent and two temporary workers. The positions were: Lab Director, Lab Scientist II (two, one permanent, one temporary), Lab Scientist I, Lab Technician II (four), Lab Technician I (two, one permanent, one temporary), Lab Assistant, and Water Sampler. One of the Lab Technician II personnel was acting laboratory supervisor.

The laboratory staff are responsible for monitoring the raw water entering the two plants for changes in levels of natural or synthetic inorganic or organic materials that might require adjustment of operating conditions, for monitoring the treated water as it leaves the plant and in the distribution system to ensure that unwanted substances are below specified levels and that parameters such as disinfectant level, fluoride, pH, etc., are in the target range, and for ensuring that the bulk chemicals used in the treatment process meet specifications. They also answer complaints about water quality from users, and provide analytical support services to process engineers during plant revisions and trouble shooting.

Some of the routine tests are now performed by plant operators, and the number of tests being done in this way is increasing. Laboratory personnel provide instruction and quality control monitoring for these measurements.

Daily, monthly, and annual reports on all measurements are required of the laboratory. In addition the laboratory must adapt and modify new analytical procedures to the special conditions encountered in the Edmonton raw water supply to meet increasing demands for more sensitive and accurate monitoring of trace levels of previously unknown or unsuspected contaminants. These new tests involve identification and quantitation at levels far lower than was previously possible, and require considerable analytical expertise by the staff in conjunction with sophisticated instrumentation.

Evaluation of Present Staff Responsibilities. The staff at the water laboratory have had to cope with a drastically increased load in recent years, especially during problem times such as spring runoff. Part of the added load has come with the introduction of chlorine dioxide in the treatment process, since development of reliable analytical methods for ClO_2 and its products has required a significant investment in staff time. Increase in work load and changes in the nature of the work required of the laboratory have created some problems. One problem is that the laboratory has not been able to keep pace with the rapid evolution of new analytical methodology. Although much high quality instrumentation has been added to the laboratory, there has not been adequate trained staff to use the instruments effectively.

As routine tests are increasingly done by on-line automatic monitors or by plant operators, the laboratory staff must change from only doing routine measurements to establishing a high level of instrument and operator performance by trouble shooting, calibrating of instruments, and quality control testing. Those analyses that require considerable analytical background and expertise, and that often require expensive and sensitive instruments, must continue to be done in the laboratory by skilled staff, but fewer routine wet chemical tests should be required of laboratory staff. Also, the laboratory staff will need to be responsible for automation and instrument-interfaced laboratory information management systems as they are introduced. Under these conditions the laboratory manager, who is responsible for providing the measurement data necessary to ensure water quality and safety, must have a strong background and training in analytical methodology and in the processing and evaluation of measurement data as well as skills in the management of a sizable staff of laboratory scientists and technicians. The manager must also maintain an in-depth understanding of new developments in analytical techniques and in laboratory operation, and apply these developments appropriately to the laboratory.

High priority should be given by management to ensuring that all workers in the laboratory identify with the goals of producing high quality data in which they can take justifiable pride. To encourage a positive attitude it is important for workers to see the significance of their measurements; this is difficult if large numbers of data are collected on parameters that do not change on a week to week basis, or on parameters that are well below levels where they are of concern. The frequency of measurements should be reviewed and modified to fit variations in raw water conditions.

Evaluation of Staffing Needs. Implementation of laboratory automation and a laboratory information management system, with the related computer interfacing of instrumentation that will be needed, will require a substantial upgrading of staff. At the present time the number of staff with sufficient analytical background and training necessary to do basic method development and trouble shooting is too low. The resulting backlog in method development and implementation has created work overloads for some of the present staff, and has affected laboratory performance through delays in solving analysis problems and through reduced quality control monitoring. Additional planning and organization is required, not only to solve the present problems, but to provide for future upgrading in automation and data handling. Staff upgrading can be accomplished partly by hiring new staff with the necessary expertise and experience, and partly by providing additional training to present staff. Outside consultants may be used to provide advice on which of the many automation and laboratory information management systems that are available would be best suited to the needs of the Edmonton laboratory, but for efficient implementation and day-to-day operation the laboratory must have in-house competence.

For the same sample workload the number of staff required to operate the laboratory should decrease with time as the extent of automation increases, but the technical competence of the individual staff members will have to increase; future manpower decisions should be made with this point in mind. Another point to be considered when hiring additional staff is that high priority should be given to the establishment of a combination of management and staff in which overall goals and job responsibilities are clearly laid out and monitored for each individual. This should help avoid problems with workers failing to identify with laboratory goals of producing high quality data.

Successful laboratory operation requires expertise in modern analytical chemistry, in conjunction with organization and planning. A Ph.D. analytical chemist in the laboratory is recommended, along with 2 or 3 laboratory scientists who are familiar with analytical instrumentation and with automation and interfacing of instruments to data storage and handling devices. Several laboratory technicians will also be required to prepare reagents, monitor and trouble shoot analytical systems, check calibrations, run test samples, and perform non-routine analyses.

CONCLUSIONS AND RECOMMENDATIONS

Evaluation of Laboratory Test Procedures and Performance

The test procedures used by the City of Edmonton Water Laboratory for physical and inorganic chemical analyses in most cases used are those recommended by recognized organizations as standard methods. An exception is the determination of chlorine dioxide and related chlorine-containing compounds in process streams and in water entering the distribution system. Results for these analyses have been unreliable. This problem has recently been recognized by the laboratory, and new methods are under development to replace past unsatisfactory ones. Evaluation of these methods to assess their accuracy and ruggedness is needed, especially the method for chlorite.

The GC purge-and-trap and head space methods, along with the UV absorbance measurements, as used provide only a general pattern of contamination by the more volatile organic substances present in the raw water. Identification and quantitation of these compounds of potential health hazard should be done. The GC methods will not detect less volatile components; their regular monitoring should be part of the laboratory analytical program.

Some analyses such as fluoride are being determined by both on-line and manual methods because the on-line procedure is not trusted. In these situations the on-line system should be brought under control so that duplication is avoided.

The reported precision of the inorganic and physical methods appears adequate, but data on regular replicate runs and control charts should be available for all analyses. Checks on analytical accuracy have been limited to recently introduced testing of operators for routine parameters, and occasional spikes. Systematic checks on analytical accuracy through analysis of standards and reference samples from outside organizations should be required of all analysts in the laboratory.

A rigorous quality assurance program should be instituted. It should cover all parts of the analytical process, from sampling through data reporting. Incorporated into the plan should be provision for regular checks on analytical accuracy through the analysis of blind external samples from the EPA, Alberta Environment, or other responsible sources.

Future Manpower and Equipment Needs of the Edmonton Water Laboratory

The Edmonton Water Laboratory has many good features, but several weaknesses. Completion of the laboratory renovations now underway will provide adequate space and analytical facilities for the near future, although the separation of the office of the laboratory supervisor from the two laboratories, and the laboratories from each other, is inefficient and hinders communication. The equipment and apparatus available in the laboratories is generally adequate, although consideration should be given to the purchase in the future of a high performance liquid chromatograph (HPLC) for the investigation of nonvolatile organic compounds, of an on-line ultraviolet absorbance spectrophotometer for upstream monitoring of humic related materials during spring runoff periods, and to an autosampler-graphite furnace accessory for the atomic absorption spectrophotometer to provide increased sensitivity for more frequent testing for trace toxic metals. Purchase of a gas chromatograph-mass spectrometer (GC-MS) is not recommended at this time. The GC-MS data needed for trace organic identification should be obtained for the near future by contract arrangements with an external commercial or government laboratory.

Major priority should be given immediately to the implementation of more on-line monitoring and automation of routine testing, and over the next two years to the development in the laboratory of more efficient data handling and reporting procedures. The laboratory is seriously deficient in these areas. Use of autosamplers, flow-through cells, and on-line sensors should be increased. A laboratory information management system should be implemented, with measurement data from the instruments fed to a central laboratory computer for processing and report preparation. The system should be implemented in steps, with the end goal of linking within the next 4 to 5 years of all measurement devices to a central data collection and processing unit. This will improve speed, efficiency, and reliability, and should reduce or eliminate the need for routine weekend and night time staffing of the analytical laboratory. As demand for new tests increases, additional automation will decrease the need for more manpower to perform the extra analyses.

To ensure maintenance of high quality, efficient analytical productivity by the laboratory, periodic audits should be performed. A frequency of once every two years is recommended, at least initially, to assess laboratory data quality, efficiency, and productivity.

Summary of Specific Recommendations

In summary, my recommendations regarding the City of Edmonton Water Laboratory operations with respect to the areas which I evaluated are as follows:

1. The laboratory should institute as soon as possible a rigorous, systematic quality assurance program to ensure that all data reported has adequate precision and accuracy. The program should include all areas, from sampling through sample custody, preanalysis handling and treatment, measurement, and data processing. It should also include a systematic monitoring of laboratory performance by analyses of external samples for which the correct values are not known in advance by the analyst. Quality control charts should be used to ensure all measurement processes are in a state of statistical control. To assess progress toward this goal a laboratory audit is recommended approximately every two years until laboratory quality warrants less frequent checking.

2. The laboratory should give immediate priority to increasing efficiency and reporting accuracy through automatic sample handling and data processing. Initially attention should be given to use of on-line monitors, flow-through measurement devices, and autosamplers. Instruments should be interfaced to a centralized laboratory information management system that stores the data in a computer and allows computerized preparation of data reports. The goal should be to have all measurement systems in the laboratory connected to a laboratory data processing center within the next two to three years.
3. To achieve the quality assurance and laboratory upgrading recommended in points 1 and 2 requires an advanced in-house level of analytical expertise. The present laboratory staff should be upgraded to increase their background in modern analytical instrumentation and data processing. This might be done by adding laboratory scientists with analytical training and experience at an advanced level to perform methods development, trouble shooting, data processing, and quality assurance monitoring, along with provision of additional training for some of the present staff. Several technical school graduates in chemical technology will continue to be needed to perform specialized tests, prepare reagents, and operate instruments and data acquisition stations.

With the implementation of the above recommendations in an orderly way over the next two to three years, the city water laboratory can be brought to a level that will ensure rapid, reliable, and accurate monitoring of all the chemical parameters necessary to provide safe, high quality water from the City of Edmonton water treatment plants.

Selected Literature on Analysis of Chlorine Dioxide and Related Species

1. Adams, D.B.; Carter, J.M.; Jackson, D.H.; & Ogleby, J.W. Determination of Trace Quantities of Chlorine, Chlorine Dioxide, Chlorites and Chloramines in Water. Proc. S.W.T.E., 15, 117 (1966).
2. Aieta, E.M.; & Roberts, P.V. Chlorine Dioxide Chemistry: Generation and Residual Analysis. Chemistry in Water Reuse. (W.J. Cooper, ed.), Ann Arbor Sci. Publ., Ann Arbor, MI (1981).
3. Aieta, E.M.; Roberts, P.V.; & Hernandez, M. Determination of Chlorine Dioxide, Chlorine, Chlorite and Chlorate in Water. Jour. AWWA, 76:1:64 (January 1984).
4. Barbolani, E.; Piccardi, G.; & Pantini, F. Potentiometric Titration of Chlorine and Its Oxy Compounds in Water. Anal. Chim. Acta, 132:233 (1981).
5. Chen, T.H. Spectrophotometric Determination of Microquantities of Chlorate, Chlorite, Hypochlorite, and Chloride in Perchlorate. Anal. Chem., 39:804 (1967).
6. Dormond-Herrera, R. & Mancy, K.H. Voltammetric Membrane Chlorine Dioxide Electrode. Anal. Letters, 13(A7):561 (1980).
7. Dressman, R.; Moore, L.; & Stevens, A. Table Comparing the DPD and Starch/Iodide Methods for Determination of Chloramine Residuals. Personal communication (1986).
8. Feuss, J.V. Problems in Determination of Chlorine Dioxide Residuals. Jour. AWWA, 56:5:607 (May 1964).
9. Gordon, G. Improved Methods of Analysis for Chlorate, Chlorite, and Hypochlorite Ions at the Sub-mg/L Level. Proc. AWWA WQTC, Nashville, Tenn. (1982).
10. Gordon, G. & Ikeda, Y. Lower Detection Units Found for Chlorine Dioxide Contaminants. Jour. AWWA, 76:98 (1984).
11. Hong, C.C. & Rapson, W.H. Analysis of Chlorine Dioxide, Chlorous Acid, Chlorite, Chlorate and Chloride in Composite Mixtures. Can. Jour. Chem., 46:2061 (1968).

12. Haller, J.F. & Listek, S.S. Determination of Chlorine Dioxide and Other Active Chlorine Compounds in Water. Anal. Chem., 20:639 (1948).

13. Harp, D.L.; Klein, R.L.; & Schoonover, D.J. Spectrophotometric Determination of Chlorine Dioxide. Jour. AWWA, 73:387 (1981).

14. Hodgden, H.W. & Ingols, R.S. Direct Colorimetric Method for Determination of Chlorine Dioxide in Water. Anal. Chem. 26:1224 (1954).

15. Masschelein, W. Spectrophotometric Determination of Chlorine Dioxide with Acid Chrome Violet K. Anal. Chem., 38:1839 (1966).

16. Milltner, R. Measurement of Chlorine Dioxide and Related Products. USEPA, EPA 1.0 M46 (1977).

17. Myhrstad, J.A. & Samdal, J.E. Behavior and Determination of Chlorine Dioxide. Jour AWWA, 61:4:205 (Apr. 1969).

18. Palin, A.T. Determination of Free and Combined Chlorine in Water by the Use of Diethyl-p-Phenylene Diamine. Jour. AWWA, 49:873 (1957).

19. Palin, A.T. Colorimetric Determination of Chlorine Dioxide in Water. Water Sewage Works, 107:457 (1960).

20. Palin, A.T. Determination in Water of Free and Combined Available Chlorine, Chlorine Dioxide and Chlorite, Bromine, Iodine, and Ozone, Using Diethyl-p-Phenylene Diamine (DPD). Jour. Inst. Water Engrs., 21:537 (1967).

21. Palin, A.T. Determining Chlorine Dioxide and Chlorite. Jour. AWWA, 62:8:483 (Aug. 1970).

22. Palin, A.T. Analytical Control of Water Disinfection with Special Reference to Differential DPD Methods for Chlorine, Chlorine Dioxide, Bromine, Iodine and Ozone. Jour. Inst. Water Engrs., 29:139 (1974).

23. Palin, A.T. Current DPD Methods for Residual Halogen Compounds and Ozone in Water. Jour. AWWA, 67:32 (1975).

24. Palin, A.T. A New DPD-Steadifac Method for Specific Determination of Free Available Chlorine in the Presence of High Monochloramine. Jour. AWWA, 72:121 (1980).

25. Palin, A.T. & Darrall, K.G. A Modified DPD Titrimetric Procedure for the Determination of Chlorine Dioxide and Chlorite in Water. Jour. Inst. Water Engrs. and Scientists, 33:5:467 (Sept. 1979).

26. Piccardi, G.; Barbolani, E.; & Pantani, F. Comparison Between Colorimetric and Electrometric Methods for Chlorine and Its Derivative Compounds. Water, Air, & Soil Poll., 13:197 (1980).

27. Post, G. & Moore, L. The Determination of Chlorine Dioxide in Treated Surface Waters. NTIS PB-215-480 (1973).

28. Post, M.A. & Moore, W.A. The Determination of Chlorine Dioxide in Treated Surface Waters. Anal. Chem., 31:1872 (1959).

29. Smart, R.B. & Freese, J.W. Measuring Chlorine Dioxide with a Rotating Voltammetric Membrane Electrode. Jour. AWWA, 74:530 (1982).

30. Standard Methods for the Examination of Water and Wastewater. APHA, AWWA, and WPCF. Washington, D.C. (15th ed., 1980). 410 Chlorine Dioxide: A. Iodometric Method; B. Amperometric Method; C. DPD Method (tentative).

31. Suzuki, K. & Gordon, G. Direct Determination of Chlorite Ion in the Presence of Excess Hypochlorite Ion. Anal. Chem., 50:1596 (1978).

32. Tang, T.-F. & Gordon, G. Quantitative Determination of Chloride, Chlorite, and Chlorate Ions in a Mixture by Successive Potentiometric Titrations. Anal. Chem., 52:1430 (1980).

33. Wheeler, G.L. et al. A Rapid Microdetermination of Chlorine Dioxide in the Presence of Active Chlorine Compounds. Microchem. Jour., 23:160 (1978).

34. Wong, G.T.F. Factors Affecting the Amperometric Determination of Trace Quantities of Total Residual Chlorine in Seawater. Envir. Sci. & Technol., 16:11 (1982).

35. Yamasaki, S.; Ohura, H.; & Nakamori, I. Borohydride Ion as a Reductant for Chlorite Ion. Bunseki Kagaku, 22:843 (1973).

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TABLE 6. THE COMPARISON OF DPD AND STARCH/IODIDE METHODS FOR THE DETERMINATION OF CHLORAMINE RESIDUALS, DOSED WITH 9.8 mg/L MONOCHLORAMINE AND USING 5.0 mg/L HUMIC ACID (SODIUM SALT)

				DPD METHOD !		
pH	Time (Hours)	Temp. (C)	S/I * (ug/L)	MCA (ug/L)	DCA (ug/L)	SUM (ug/L)
5.5	24	6	8.8	3.6	5.4	9.0
5.5	72	6	8.3	1.8	7.0	8.8
5.5	144	6	8.5	0.8	8.1	8.9
7.5	24	6	8.8	7.5	1.3	8.8
7.5	72	6	7.8	5.9	2.2	8.1
7.5	144	6	6.6	4.6	2.7	7.3
11.5	24	6	9.1	9.3	0.1	9.4
11.5	72	6	8.8	9.2	0.0	9.2
11.5	144	6	9.0	9.4	0.1	9.5
5.5	5	20	8.5	6.0	2.8	8.8
5.5	24	20	8.3	2.8	6.0	8.8
5.5	72	20	7.4	1.1	6.5	7.6
5.5	144	20	6.6	0.5	6.4	6.9
7.5	5	20	8.6	8.4	0.8	9.2
7.5	24	20	7.4	6.7	1.0	7.7
7.5	72	20	4.9	4.5	0.7	5.2
7.5	144	20	2.5	2.1	0.3	2.4
11.5	5	20	8.9	9.4	0.0	9.4
11.5	24	20	8.5	9.0	0.1	9.1
11.5	72	20	8.6	9.0	0.0	9.0
11.5	144	20	8.0	8.5	0.1	8.5
5.5	5	36	8.0	6.0	2.6	8.6
5.5	24	36	6.0	2.3	3.7	6.0
5.5	72	36	1.8	0.5	1.7	2.2
5.5	144	36	0.4	0.2	0.5	0.7
7.5	5	36	7.8	8.6	0.2	8.8
7.5	24	36	6.0	6.6	0.1	6.7
7.5	72	36	4.2	4.4	0.1	4.5
7.5	144	36	2.9	3.2	0.1	3.3
11.5	5	36	8.5	9.1	0.0	9.1
11.5	24	36	7.8	8.6	0.1	8.7
11.5	72	36	7.6	7.9	0.0	7.9
11.5	144	36	6.6	7.0	<0.1	7.0

* S/I = Starch/Iodide method for total oxidant residual (as Cl)

! MCA = Monochloramine residual (as Cl)
DCA = Dichloramine residual (as Cl)
SUM = MCA + DCA (as Cl)

EVALUATION OF MONITORING AND TREATMENT FOR PROTOZOAN PATHOGENS

EDMONTON WATER SUPPLY EVALUATION

Report presented to

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and the

City of Edmonton

by

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INTRODUCTION

This report is the result of a three-day site visit in Edmonton on April 2-4, 1986. The primary objectives of this visit were to evaluate monitoring for protozoan pathogens at Edmonton, to evaluate treatment methods with regard to protozoan pathogen removal, and to make appropriate recommendations (Tasks 3.2.1 and 4.3). A secondary objective was to evaluate information on asbestos.

EDMONTON SITE VISIT

On April 1, I flew to Edmonton, Alberta, to participate in a comprehensive evaluation of the water supply of Edmonton. At Edmonton I met with the steering committee for the water supply study on April 2.

On the afternoon of April 2, I visited the E. L. Smith plant. This is the newer facility, and is located upstream of Edmonton in a rural area. The plant was opened in 1976 and later expanded. The plant employs alum coagulation at pH 7 and upflow clarification, followed by upflow lime softening at a pH of about 9.5, recarbonation to a pH of about 8.0, and filtration. This plant can attain filtered water turbidities close to 0.10 NTU. The engineer in charge, Karim Kassam, seemed very conscientious. He is a registered engineer (P. Eng.) and is continuing his schooling at the University of Alberta in environmental engineering.

On April 3, I spent the morning at the Rossdale plant complex. This facility has two treatment trains consisting of alum coagulation and settling, lime softening and settling, recarbonation and settling, and filtration. A third treatment train consists of two upflow clarifiers. In this respect, it has some similarity to the E. L. Smith plant. Total capacity of the Rossdale plants exceeds capacity of the E. L. Smith plant. That afternoon I went to the University of Alberta.

On April 4, I spent the entire day at the Rossdale treatment plant. I again reviewed physical facilities (mixing, flocculating, and chlorine addition) and obtained data on plant operations. I spent much of the day talking to George Walker and Myron Lahola. My impression was that they sincerely want to do a good job of water treatment. Their task is made quite difficult, at times, by constraints of staff and equipment.

COMMENTS ON WATER TREATMENT

E. L. Smith Plant

The E. L. Smith plant appeared to be well operated but a few problems were noted. Mr. Kassam needs additional staff to perform jar tests. The available staff are so busy operating the plant that they have little free time to do exploratory work or optimization studies. Operator education is a need at this plant. The mixing zone in the lime softening upflow clarifier displayed uneven hydraulics. One side was turbulent, with milky water. A portion of the other side of the mixing zone had relatively clean and still water. Mixing should be even throughout, I think. Perhaps the discharge from the clarifier was greater on the turbulent side, or perhaps the shape or presence/absence of baffles was causing a flow distribution problem. The operators, however, thought that when the flow pattern was uneven the unit performed better. This seems to contradict engineering concepts. I think this is evidence of a problem that needed investigation, and of a need for more operator training. I think a greater degree of curiosity, a tendency to question the circumstances, should be displayed.

Rossdale Intake, Raw Water Quality, and Disinfection Practice

The location of the Rossdale plant is very unfortunate, from a water treatment perspective. Storm sewers discharge to the North Saskatchewan River upstream from the water works intake. I am surprised that the engineers responsible for this plant did not consider the effects of sewage and storm water discharge and locate the plant upstream, away from the City (as the Smith plant is now located). Cincinnati went upstream beyond urban waste discharges in the early 1900's, when the water treatment plant was built. Obtaining the best quality of source water is fundamental, yet this was not done at Rossdale. This is an obvious flaw in the system. Rainfall washes road oils off Edmonton's streets and into the drinking water. The potential for microbial contamination is present also. Storm water runoff has been reported in the literature to be a source of coliform organisms for two decades or more. Also, some sanitary sewer connections may have been made in the storm sewer system. Discharges from the storm sewers to the North Saskatchewan River would be expected to lower the quality of the river water.

The location of the Rossdale plant raw water intake is not in accord with preferred sanitary engineering practice. "Recommended Standards for Water Works," a guidance document prepared by sanitary engineers of the ten Great Lakes Upper Mississippi River Basin States, plus Ontario, states (p. 12), "Each water supply should take its raw water from the best available source which is economically reasonable and technically possible." The USPHS's "Manual for Evaluating Public Drinking Water Supplies," also deals with intake location (p. 24). It states, "The purpose of the plant intake is to withdraw continuously adequate quantities of the best available grade of raw water. When selecting the intake location, the stream or lake bottom character, currents, and potential sources of pollution must be considered." These documents support the recommendation that the intake be moved upstream to obtain better raw water.

The discharge of street runoff and some untreated domestic sewage (via cross connections) is a serious problem. For twenty years or more, storm water runoff has been documented as highly polluted. The storm water runoff would contain not only microbial contaminants associated with surface water runoff, but also some organisms found in raw sewage, because of illegal or improper connections of some sanitary sewer lines from homes. The North Saskatchewan River at the Rossdale intakes has to be considered polluted. Fecal coliforms in the raw water at Rossdale averaged 630 per 100 mL (geometric mean) in February, 1986. During the same time interval, the fecal coliforms in raw water at the E. L. Smith plant averaged less than 4 per 100 mL. These results suggest that sewage discharges are contaminating the river between the E. L. Smith intake and the Rossdale intakes.

During the late February-early March snowmelt and runoff episode, fecal coliforms at the E. L. Smith intake were on the order of 10 to 200 per 100 mL. At Rossdale, fecal coliforms in the raw water were in the range of 200 to 4000 per 100 mL. The report of the February-March episode indicates that chlorine dioxide residual was not maintained at any point of measurement at Rossdale for some unspecified time period. To quote from the report by Edmonton Water and Sanitation, "Also on Thursday afternoon it was decided to move the feed point from the raw water to post clarification at Rossdale as soon as possible since the dosage had to be so low to avoid exceeding the 0.5 total oxidant level that there was no longer a measureable residual anywhere in the process." If no measureable ClO_2 residual existed anywhere in the process, according to the Utility, how could those in charge have assurance that effective disinfection was being accomplished? This statement from the utility report raises the question of whether or not disinfection was really in control on that Thursday afternoon. This was at a time when fecal coliforms in the raw water at Rossdale ranged from 200 to 2000 per 100 mL.

With no residual chlorine dioxide present, the disinfection burden would be on the chloramine. Because waterborne pathogenic viruses are shed by humans, and fecal coliforms could come from humans, viruses could be in the raw water at Rossdale from time to time. Berman and Hoff reported that 99 percent inactivation of simian rotavirus (SA 11) was attained in 6 to 7 hours with a 10 mg/L residual of preformed chloramine at pH 8 and 5°C. Their work suggests that reliance on chloramine to disinfect a contaminated surface water is not prudent. Use of a weak disinfectant to treat a very clean, uncontaminated surface water may present fewer problems.

Because chloramine is an inferior disinfectant, with respect to its ability to inactivate microorganisms, I would not recommend treatment of contaminated water with chloramine. In actual practice, preformed chloramine is seldom used, so free chlorine contacts the water and the organisms for varying amounts of time before chloramine forms. The short contact time with chlorine may, in some cases, be as beneficial as the much longer contact with chloramine. At Edmonton, ammonia is added to raw water, and chlorine is added later in treatment. The addition of chlorine to settled water in the stilling basin (nearly total absence of mixing here) makes analysis of the disinfection at Rossdale extremely difficult. It is practically impossible to know what amounts of chloramine or chlorine microorganisms would be exposed to before the water leaves the stilling basin and mixes thoroughly, just before it enters the filters.

While I visited the Rossdale plant, I recommended that the chlorine addition point be moved to the turbulent zone ahead of the stilling basin. The addition of chlorine just ahead of recarbonation would achieve better mixing of the chlorine and faster formation of chloramine. It would, however, result in a brief period of free chlorine at high pH, where OCl^- would be the predominant agent, rather than HOCl. OCl^- is not a very effective agent, although it is a better disinfectant than chloramine.

I think long-term reliance on chloramine disinfection at Rossdale is not prudent. A disinfection practice better able to inactivate the pathogens to be expected in the poor quality raw water at Rossdale would be the use of chlorine dioxide as a disinfectant. It is highly effective against viruses and Giardia cysts. If a chlorine dioxide residual can not be maintained to provide disinfection, then the plant should be switched to free chlorine disinfection. If, for health effects reasons, chlorine dioxide use is discontinued, chloramine should not be used as the primary disinfectant. Use of a weak disinfectant invites acute waterborne disease problems. Alternatives to chloramine should be considered.

Although extrapolation of disinfection experimental results involves some uncertainty, available results do give a limited amount of guidance. Research with Giardia muris cysts indicates that 99% inactivation of G. muris is obtained with residual concentration - contact time (C•t) products (mg/L • minutes) in a range 7.2 mg/L • minutes to 18 mg/L • minutes, for pH 7, 5°C. No pH 9, 5°C data are available yet. Results at 25°C indicate that C•t products are lower at pH 9 than at pH 7. If the pH effect at 5°C is similar to that at 25°C, the C•t values of 7 to 18 mg/L • minutes should be useful for evaluating chlorine dioxide disinfection at Edmonton. If the ClO_2 residual is 0.1 mg/L, the time of contact should be 70 to 180 minutes to yield a C•t product of 7 to 18 mg/L • minutes. If the ClO_2 residual is 0.2 mg/L, the corresponding contact times needed would be 35 to 90 minutes. These valuesare tentative, and should be re-evaluated upon completion of research presently under way at Ohio State University

(Dr. A. J. Rubin) under the sponsorship of the U.S. EPA. The chlorine dioxide residuals and times given above are related to 99% inactivation. C·t values for other degrees of inactivation would be different. Chlorine dioxide application should be done in a way that does not violate any guidance given by the Canadian provincial or national agencies on this issue.

Plant Operation and Control

The staff I met and spoke with at length, mainly engineers and scientists, seemed very conscientious and concerned about doing a good job. I think that in some instances the staff lack the tools needed to do the job and should be provided with them, or should look for more imaginative ways to use the tools available.

Both treatment plants use aluminum sulfate as the coagulant in the first stage of treatment. I would recommend studying use of iron instead of aluminum, and if jar tests and pilot plant studies show iron is as effective as alum, iron should be used instead. Meanwhile, as a check on the use of aluminum in treatment, residual aluminum in filtered water and distributed water should be measured. This would not need to be a highly precise measurement to be useful to plant engineers and chemists. A Hach spectrophotometer is available, and aluminum analysis can be done with Hach equipment. I recommend doing the aluminum analysis with the Hach instrument, and checking the results by sending samples to the University of Alberta for AA analysis. Hach instrumentation was used to measure aluminum in treated water in a project at Cayuga, N.Y. The lower limit of detection was reported to be 0.02 mg/L. Measurement of aluminum would be very helpful. Aluminum that dissolves in the softening step and is carried out into the distribution system could precipitate on pipe walls.

A thin, rippled layer of aluminum on the pipe wall could reduce the hydraulic capacity of the pipe. Monitoring aluminum in filtered water will indicate to operators whether some of the aluminum added as a coagulant chemical is failing to precipitate, or is re-dissolving because of the lime softening.

While I was at the Rossdale plant, I asked some questions about plant operation.

Q. How much powdered activated carbon can be added without penetration of the filter by PAC?

A. 20-25 mg/L of PAC could cause black looking water on the filters.

Q. What strategy is used to make decisions on how to coagulate?

A. No provision exists for pH control. Jar tests can be done.

Q. What is the goal for turbidity of settled water in the alum coagulation process?

A. No goal for settled water turbidity has been set for the first stage of coagulation.

Q. What is the goal for turbidity applied to the filters?

A. No goal has been set.

Q. What is done to control turbidity going to the filters?

A. No particular action is taken.

In my opinion, more effort ought to be directed to producing low turbidity water, and to acting upon the data that are developed during routine plant monitoring activites. Present procedures seem to place a greater emphasis on monitoring and lesser emphasis on taking actions based on the monitoring.

Continuous monitoring of filtered water turbidity is practiced. This is a valuable practice. Continuous monitoring can provide early information as treated water turbidity changes. If filters are backwashed before turbidity rises excessively, breakthrough of particulate matter can be minimized.

Calibration of the continuous flow Hach turbidimeters should be done on a daily basis by collecting a grab sample and measuring the turbidity on a bench model turbidimeter, and then adjusting the continuous flow meter to give the same reading. This should be done promptly and during times of stable water quality, so that filtered water turbidity does not change between the time of sample collection and meter adjustment.

Another aspect of plant operation and control that should be modified somewhat is jar testing. The published work on jar testing often recommends that these tests should be performed at raw water temperature instead of room temperature. To accomplish this, the water utility shop workers should build clear plexiglass boxes for each jar test apparatus. The boxes can serve as water baths when raw water is circulated through and drained to waste. Raw water from the raw water tap in the plant laboratory can thus serve as the coolant for the water bath. A refrigeration unit would not be needed if raw water flow is sufficient to avoid significant temperature increase within the building.

Another helpful aspect in performing jar tests is to add chemical through a funnel, at the tip of the jar test mixer blades. This would be a mode of chemical addition similar to that practiced for Rossdale No. 2 at the present time. Pouring the chemicals onto the water surface is not as effective in jar tests, and is not recommended for full scale coagulation practice either. Jar tests should be performed in a way that approximates actual treatment practice as closely as possible. This will be easier to do at Rossdale, where coagulation and settling are done in the conventional clarifier manner. At E. L. Smith, use of upflow clarifiers will make using jar tests as a predictor plant behavior more difficult, but extended research with jar tests in the laboratory may enable plant staff to determine those jar test operating conditions that yield results most closely approximating the full scale plant.

If lime softening jar tests are also performed, I recommend that settled water from the softening test be filtered. Experiments should be conducted to determine whether filter paper filtration or sand column filtration more closely approximates the turbidity attained in the full scale plant. Although several weeks or even a few months of work might be needed to discover the procedures that best agree with full scale plant performance, and then to verify that these procedures are valid over a range of raw water quality conditions, if this can be done, plant staff will have confidence that a rapid method for evaluating treatment is available. This would be very useful when raw water quality suddenly changes and the chemical conditioning that was formerly effective no longer works.

The problem of sudden water quality changes and loss of control of water quality in the plant is not merely hypothetical. An example of temporary loss of control of treated water quality is the runoff episode of late February - early March, 1986. At Rossdale No. 1, filtered water turbidity was 0.35 NTU at 2200 hours on March 1. It rose to 1.5 NTU at 0200 hours on March 2 and peaked at 2.1 NTU at 1200 hours. At 1600 hours it had dropped below 1 NTU (0.85 NTU), and never went above 1.1 NTU the remainder of the day.

If the utility had a group responsible for treatment studies and process evaluation, jar tests and treatment evaluations could be conducted throughout the year. During times when treatment was likely to be difficult, jar test studies and other work could be conducted at a more intense level. When problems were observed in the jar tests, studies could start immediately to define appropriate chemical doses to treat the water satisfactorily. This would be much better than discovering that all of the basins in the treatment plant contained water that was not likely to meet the objective range for turbidity after filtration. If episodes of poor water quality are associated with events such as snowmelt or thunderstorm runoff, the staff should begin treatment studies in anticipation of bad water, based on occurrence of events that usually cause raw water to be difficult to treat.

Some staff should be employed for the purpose of treatment process research and treatment chemical evaluation. At the Cincinnati Water Works, several people do this. Population served by the utility here would be slightly less than one million. For Edmonton, a good combination would be an engineer, a chemist, and a technician, with the understanding that everyone was part of a team, and the engineer and chemist would do laboratory work as needed. Expertise in treatment is gained by working with the water in question. Thus, formation of a special group for this purpose is recommended.

In addition to a possible shortage of staff to evalute water treatment processes and chemicals, the lack of pilot facilities is a severe handicap. Process changes, such as changes in disinfectant chemical or point of application, or a change from aluminum to iron metallic coagulant, should be explored on a small scale before work is done full scale. A logical first step would be to conduct jar tests in series, first coagulating and settling and then softening and settling, and then recarbonating, settling, and filtering. Such tests can be done quickly and relatively easily. A variety of treatment options can be checked in a short time, e.g., a range of coagulant doses. The most successful chemical combinations should then be evaluated in a pilot plant, to confirm the jar test results.

The wide variation of raw water turbidity, ranging from 10 NTU or lower in the winter to as much as 250 NTU during episodes of high turbidity, makes treatment of the source water difficult. Although the time consumed by pilot plant studies might make it difficult to use a pilot facility to get near-term answers for an episode only a few days in duration, the experience gained during a short period of bad water quality might be useful when such a problem was repeated. Using a pilot plant to explore treatment options during periods of high turbidity could be a learning experience for the staff and could improve their understanding of how to cope with poor water quality in the future.

The pilot plant used by the University of Alberta at the Rossdale facility is a very fine plant. Its capability does not include lime softening, however, so the staff are unable to fully evaluate treatment of the North Saskatchewan River in the Rossdale manner. If the water utility obtains the pilot plant built by the University of Alberta, softening and recarbonation should be added. If the utility builds its own pilot plant, the process steps used in the full scale plant should be included in the pilot facility. I'm not sure that it is necessary to duplicate process hydraulics. The upflow clarifier step might be simulated, with the mixing and floc blanket treatment in a deep cylinder rather than in an inverted cone or pyramid.

If a complete pilot plant can not be assembled in the near future, at least some pilot filter columns should be installed at each plant. Filter columns that use settled water could be operated at a higher rate than the rate used for filters in the plant. This would enable plant operators to have some indication of how filters were going to behave, especially at times of rapid quality change. A concept that could be explored is filtration of coagulated, settled, unsoftened water. How would filtered water quality from the plant compare to water filtered before softening? Perhaps this approach could allow the operators to gain some time in their attempt to monitor quality and discover problems earlier in the treatment process. For the long run, however, availability of a complete pilot plant would be advantageous, because then studies of coagulation could be conducted.

It is highly important that provisions be made for performing tests on the raw water to evaluate treatment changes being contemplated. The practice of implementing changes full-scale with limited exploration by jar test and no pilot plant verification is risky. Confirmation of preliminary results is being made full-scale with water produced and distributed to the customers. For minor operational changes such as addition of PAC or modification of alum dose, this would be considered normal practice. Major changes, such as use of chlorine dioxide or substitution of iron for alum, ought to be piloted unless some other base of experience (such as a nearby treatment plant using the process under consideration) exists. The latter situation is highly unlikely for Edmonton, so a pilot plant is almost a necessity.

Training and Education for Treatment Plant Staff

Training, education, and professional development for the water treatment plant staff should be encouraged by water utility management. Because

Edmonton is a long distance from the populous Eastern part of Canada, and because only a small number of large cities in Western Canada have water filtration plants, the opportunities for person-to-person contact and plant visits are minimal for Edmonton's treatment plant operators. Increased efforts should be made to provide the needed training. Resources of the various professional associations active in water supply could be helpful, but if more training is needed than others provide, Edmonton should take the necessary action to provide the training.

The expenditure of funds for a pilot plant would provide Edmonton with a valuable operator training facility. Pilot plants can be operated as a training tool, enabling operators to see how changes in chemical dose cause changes in effluent quality. Operator training should also involve community college studies. Some university courses might be appropriate, and University of Alberta faculty might be employed to conduct training involving use of the pilot plant. The numerous professional development opportunities related to attending water works meetings should not be overlooked. The American Water Works Association schedules a meeting on water quality each November or December. Technical topics on water chemistry and water microbiology are presented. Also, the AWWA Annual Conferences in June, with Sunday Seminars on selected topics, offer another learning opportunity, especially for water treatment plant engineers. Canadian professional association activities that relate to water supply should be considered essential for key water utility staff members. Peer contacts and discussions serve to bring new ideas to the plant staff. A well trained staff should be able to operate Edmonton's Water treatment plants more effectively and more economically. Every effort should be made by management to pay salaries commensurate with the expertise of the staff, in order to retain capable and motivated employees. I did not ask for specific information on salaries, but my experience elsewhere indicates that often they are too low. Low salaries are counter productive in the long run.

GIARDIA CYSTS AND GIARDIASIS

Giardiasis Outbreak, December, 1982 - April, 1983

During January through March, 1983, about 550 cases of giardiasis were reported in Edmonton. This apparently caused a great deal of concern, because the outbreak was studied by the Edmonton Local Board of Health, Alberta Environment under took a *Giardia* monitoring program, I was contacted by George Walker and asked to give an opinion on removal capabilities of softening and sedimentation, and eventually Ed Lippy was brought in to evaluate the Rossdale facilities. At this point in time proving or disproving that the giardiasis outbreak was waterborne would be impossible. Nevertheless, some misconceptions may have occurred and thus should be discussed.

My letter, dated March 30, 1983, to George Walker was based on a telephone conversation he and I had. In my letter I stated that if 80% cyst removal could be attained in each settling step and at the filters, removal would be $1 - [0.2]^3$ or 99.2%. I also stated that 90% removal in each of three solids separation steps would yield an overall removal of 99.9%. Since March 30, 1983, I have had the opportunity to evaluate *Giardia* cyst removal by sedimentation and have had the opportunity to visit Edmonton. On the basis of both of these experiences, I would be inclined to revise my estimates of *Giardia* cyst removal at Edmonton.

If the Rossdale plants were treating a low turbidity water late in 1982 and early in 1983 (likely because of below-freezing conditions), I would not expect that turbidity reduction by alum coagulation and settling would be 80% or 90%. Turbidity data for the various plant processes were not available in the 1983 records. The February-March, 1986, turbidity data show that turbidity reduction in the alum treatment step at Rossdale may vary from 0% to 90%. Our pilot plant research showed that cyst removal and turbidity removal were similar. The data from early March, 1986, show that often the turbidity in the stilling basin was nearly as high as the turbidity of the settled water after alum coagulation. When low pH lime softening is employed, the calcium carbonate precipitate does not form a gelatinous floc. Removal of particulate matter would not be as effective when high pH lime softening is used to remove calcium carbonate and magnesium hydroxide. The March 30, 1983 estimate that 80% or 90% removal of cysts would occur in the first stage of treatment and again in settling after lime softening was probably excessively high. Actual removal of cysts in the two stages of sedimentation could be considerably below the 96% to 99% that would be obtained by 80% to 90% efficiencies in each basin. Thus, the overall cyst removal efficiency for the plant as estimated in the March 30, 1983 letter could be lower than the 99.2% to 99.9%, unless the filtration cyst removal efficiency was greater than 90%. My estimate of 99.2% to 99.9% may have been too high. Ultimately, the quality of the filtered water is what is important, but it can be influenced by the quality of the water applied to the filters.

After the outbreak occurred, monitoring for Giardia cysts was begun. Unfortunately, some of the monitoring was done with 7 micron wound element filters. Current practice for wound filters is to use a nominal 1 micron pore size. Failure to find Giardia cysts on the 7 micron wound element filter is not surprising. I discussed the sampling with Joe Milos and he informed me that polycarbonate membrane filters were used for much of the treatment plant sampling. This generally has not been done in other treatment plants, but is the technique used for pilot plant research. I have more confidence in the membrane filter approach, where sufficient volumes of water can be sampled. Thus, the results from December 7, 1983 to May 9, 1984 indicate that Giardia cysts were not detected. These results, however, tell us nothing about the time when the outbreak took place.

Recent work in the USA has included sampling of source waters, raw and treated sewage; and combined sewer overflow by means of 1 micron wound filters. The results have not been published, but I can inform you that cysts were found in combined sewer overflow as well as raw and treated sewage. Cysts detected in the overflow were on the order of 7/liter. Correcting for sampling efficiency, the actual number may have been 70/liter. The sewer just upstream from the Groat Bridge could be a source of these cysts. This is another factor that favors relocation of the intake upstream of the present location.

I have reviewed "Giardiasis in Edmonton," prepared by Collier and Macdonald. The authors (p. 3) discuss the number of cysts needed to ensure infection in all subjects, citing Rendtorff and stating 100 cysts are needed. This might be correct, but we do not know. Rendtorff had no knowledge on the percentage of viable cysts in the preparations he worked with, so establishing the dose needed to cause infection is difficult. The minimum infectious dose is probably important in the Edmonton situation, because the number of cases reported was on the order of 0.1 percent of the population of Edmonton. If a massive dose of viable cysts had been distributed throughout Edmonton via the drinking water, we would have expected a much larger number of cases.

With respect to the low numbers of cysts found in water supplies and the problem of waterborne outbreaks, the cyst dose needed to infect people may be fewer than 10, perhaps as low as 1 cyst. Also, cyst recovery procedures are not very efficient, and unless data are corrected for the losses that occur, cyst numbers in water will seem low.

On p. 11 Collier and Macdonald are uncertain that the decreasing incidence of giardiasis, as distance from the Rossdale plant increases, should be attributed to inactivation of cysts by chloramine. However, research has shown that chloramine can inactivate Giardia cysts, although values of disinfectant residual concentration (C) multiplied by contact time (t) needed for a 99% kill are rather high. The C·t product for a 99% kill by chloramine would be about 400 to 600 mg/L · minutes. This is the equivalent of about 7 to 10 hours for 1 mg/L and 3-1/2 to 5 hours at 2 mg/L residual disinfectant. Although it would act slowly, I'd expect chloramine to inactivate Giardia cysts if the above conditions could be attained.

On p. 28 the report discusses the clustering of cases downtown, and states that all census tract areas with incidence rates of 300 or more per 100,000 population lie in close proximity to the Rossdale Water Treatment Plant. To me, this looks suspicious, but the authors dismiss the possibility because areas with low rates of reported illness were also serviced by Rossdale, and because no clustering of cases was noted around the E. L. Smith Plant, even though there was no discernable difference in the quality of treated water at the two plants. The authors gave no data to substantiate their claim about the similarities of water quality. The important question is the actual water quality, rather than meeting a national quality goal. Studies in Colorado have shown that finished drinking water can meet the 1 NTU limit for turbidity and the total coliform limit, but giardiasis outbreaks can occur. Giardia cysts can be present in 1 NTU water, and they can survive disinfection at a level that is adequate to kill coliform bacteria.

The calculation of the number of infected people or animals needed to cause 548 cases is interesting. It is based on a particular set of assumptions, including the uniform distribution of 10 cysts/L in all treated water. Because not all Edmonton residents were ill, it is not necessary to assume that there were 10 cysts in each liter of water. This causes the cyst estimate to be high. The opposite approach, having only 10 cysts per infection, would require just 5,500 cysts to be present in the treated water, or 55,000 in the raw water, with 90 percent removal. This is not a likely situation, but it also is unlikely that 10 cysts had to be in each liter of treated water to cause the outbreak. A reasonable answer would be between the extremes.

The concentration of cysts in source water would not need to be uniform on a day-to-day basis. If Giardia cysts from dogs can infect humans, as some believe, cysts in dog feces could be washed into the North Saskatchewan River via the storm sewers upstream from Rossdale. The runoff periods would be related to winter rainfall or snowmelt. Cold cysts would survive longer than warm cysts, so accumulation of cysts on land could occur in winter. Passage of cysts in treated water might occur from time to time, rather than on a daily basis, but the intermittent discharges might have been sufficient to cause an outbreak.

Because this outbreak occurred in 1983, we can only provide opinions of what might have happened. A waterborne outbreak can not be shown to have happened, but neither can it be positively discounted.

Monitoring for Cysts

Water quality monitoring is necessary if a data base is to be developed for future use. Sampling and analyzing for Giardia cysts can be time-consuming tasks, especially the analysis. The results must be viewed in the proper context. Failure to find Giardia cysts in a water sample may or may not mean that cysts were in the sample. When raw water samples are examined microscopically, the debris collected on the filter can sometimes cover or mask the cysts. Cysts that are present can be overlooked. Even if a water sample had no cysts present, another sample of water taken later might be contaminated. The failure to find cysts in raw water should not be interpreted to mean that the source will always be free of cysts. This is especially important when sources of contamination are located upstream. Because of the very long delay between sample collection and availability of data on cyst concentration, the treatment plant operating strategy should be based on the expectation that the raw water could be contaminated at any time. Analysis of treated water samples for Giardia cysts can provide a useful record of water quality, but again because of the time involved, cyst analysis would not be useful for control of treatment processes. When filtered water samples are collected, they should be taken from individual filters. Filtered water turbidity data should be obtained when samples are collected. Monitoring the quality of water from individual filters, rather than the quality of water from the clearwell or detention basin, can provide data that can be used to evaluate treatment efficacy. After water from several filters becomes blended, variations from filter to filter can't be detected, and departures from the desired operating range are more likely to happen.

MISCELLANEOUS ISSUES

Asbestos in Water

I have been asked to comment about the possibility of asbestos-cement water mains becoming a source of asbestos fibers in drinking water. The Edmonton goal for the Aggressiveness Index (AI) is a range of 11.5 to 13. February, 1986 operating data show the treated water quality met that goal at all plants. When a water tends to deposit $CaCO_3$, or when it is balanced between dissolution and deposition of $CaCO_3$, the tendency to leach calcium from the pipe wall, to weaken the concrete in A-C pipe, and to remove asbestos fibers from the wall would be reduced. I would not expect a problem with asbestos fibers to occur at Edmonton.

One aspect of water quality related A-C pipes should be considered even by water utilities that have waters stable with respect to $CaCO_3$ saturation. Whenever A-C pipe is tapped, the cuttings should be flushed out of the pipe. A-C pipe fragments should not be allowed to fall inside the pipe, where they could be stirred up by high flows. It is my understanding that present tapping of A-C pipe is done under pressure so cuttings are flushed out. Sampling by means of fire hydrants has resulted in asbestos fiber counts 100 times higher than sampling from household taps. Good housekeeping practices in the distribution system are important for maintenance of good water quality in systems with A-C pipes, as with other systems, and should be continued.

Home Water Purifiers

When a public drinking water supply is of poor quality, or when customers customers think the water is of poor quality, some people who are dissatisfied with the water provided by the utility decide to turn to home treatment devices. Serious taste and odor problems can be a motivating factor in installation of point-of-use treatment devices. Research in the Microbiological Treatment Branch has shown that heterotrophic plate count bacteria can colonize point-of-use devices that contain granular activated carbon filters. Studies done to date have not shown that pathogenic bacteria colonize in point-of-use GAC filters.

One serious problem associated with home water treatment devices is maintenance. The home owner generally purchases the device and is responsible for its maintenance. If a point-of-use device is installed because of concerns related to health and the safety of drinking water, the home owner probably has no means of evaluating its peformance. For example, a GAC column might be installed to remove hydrocarbons, but the home owner would have no way to determine when breakthrough had occurred, except to detect tastes or odors.

The circumstance that seems most appropriate for point-of-use water treatment is the very small utility, or the individual home water supply, with installation and maintenance of the units being managed by a public agency. This takes responsibility for maintenance out of the hands of the home owner. I do not think point-of-use treatment devices are appropriate in Edmonton for controlling tastes and odors as well as organic compounds that may have adverse health effects, except perhaps as a short term response by concerned home owners. Installation of GAC treatment or development of an effective method to use PAC at the water treatment plants would be preferred. Water treatment should be done by professionals whenever possible.

If others who have been involved in the Edmonton study have recommended additional activated carbon treatment, perhaps the water utility can use such a recommendation to develop support within the local community for the construction and operation of carbon treatment. If a large majority of Edmonton residents were to install GAC point-of-use filters to control tastes and odors, it could become very difficult to build a broad base of support for centralized activated carbon treatment. People who have spent hundreds of dollars for a home device may be reluctant to pay the higher water bills needed to operate and amortize a central treatment facility. Thus, home treatment devices ultimately could have some influence on the technical, social, and economic decisions made about water treatment at Edmonton.

SPECIFIC RECOMMENDATIONS

1. Move the raw water intake for the Rossdale plant to an upstream location where the raw water is not contaminated by storm sewers or improperly connected sanitary sewers in the City of Edmonton.
2. A chemical disinfectant stronger than chloramine should be used, especially at the Rossdale plant. Because a chlorine dioxide residual has not been maintained during all raw water quality conditions, alternatives to present practice need to be considered.
3. Because lime softening is practiced at both plants, use of an iron salt coagulant should be evaluated. The treatment plants are operated beyond the optimum pH range for alum coagulation.
4. While alum is used for coagulation, aluminum in finished water should be monitored as a means of process evaluation and control.
5. Treatment plant staff should make a more intense effort to minimize filtered water turbidity at the Rossdale plant. The AWWA Quality Goal of 0.1 NTU is a good goal to try to attain, as much of the time as possible. When this goal is met, filters

should be backwashed when filtered water turbidity exceeds 0.2 NTU. If operating results show that such a goal can not be attained when it is attempted, studies should be undertaken to determine what needs to be done in order to attain the goal.

6. Plant staff should follow up on monitoring data that are collected, by modifying plant operation, as needed, to improve the quality of the filtered water. Feed back of water quality information from the laboratory to those in charge of plant operation should be used as decisions about treatment are made.

7. Jar test procedures should be improved, especially in regard to using a water bath to maintain cold water for jar tests in the winter.

8. Additional staff members should be hired so that a continuing effort in treatment evaluation, jar testing, pilot studies, and chemical evaluation can be maintained.

9. Pilot plant capability should be developed, so that staff have the tools necessary for evaluation of treatment alternatives. Major treatment changes should not be done at the plants on a full scale basis without experimental (probably pilot plant) data that show they should succeed.

10. Training for professional and technical staff should be increased to compensate for their relative isolation from their peers at other water treatment plants that coagulate, soften, and filter surface waters.

11. Monitoring for Giardia cysts should continue. Absence of cysts in raw water should not be interpreted as assuring that cysts will never be present in raw water.

12. Plant staff should attempt to maintain effective filtration and disinfection barriers at all times, to prevent passage of viruses and cysts, if they were in the raw water. Specifically, a cysticidal or virucidal dose of disinfectant should be maintained and filtration practice should be conducted, in order to attain at least a four log reduction of viruses and a three log reduction of Giardia cysts. The potential reductions could be even greater, when both processes are working effectively. For example, filtration that produces 0.1 NTU effluent with 10 NTU raw water would be expected to remove 99% of Giardia cysts and 99.9% of viruses, or perhaps more. Chlorine dioxide conditions (C·t of 7 to 18 mg/L · minutes) should give 99% inactivation of Giardia cysts at 5°C. C·t values for 99% inactivation of poliovirus 1 by chlorine dioxide have been reported in the range of 0.2 to 6.7 mg/L · minutes at pH 7, 5°C. Those data are, however, based upon ClO_2 concentrations ranging from 0.4 to 14.3 mg/L, with no data available for ClO_2 doses in the 0.1 to 0.2 mg/L range. The data suggest that conditions for Giardia inactivation must be more rigorous than conditins for poliovirus 1 inactivation. Under conditions stated above, the barriers should be effective and yield the three log cyst and four log virus reduction. When problems occur with either filtration or disinfection, to maintain the same level of organism reduction through the plant, in theory the reduced efficacy of the problem barrier should be compensated for by increased efficacy in the effective barrier. In actual practice this may be difficult, because some raw water quality problems may adversely impact both the disinfection and filtration barriers.

13. The water utility should continue to practice good maintenance procedures in the distribution system, so that the contribution of asbestos fibers from A-C pipe to the drinking water is minimized.

14. The water utility should maintain the highest practical, attainable water quality, especially with regard to taste and odor, in order to minimize the incentive of water utility customers to purchase point-of-use treatment devices. Widespread personal investment in point-of-use treatment devices could erode the broad base of support for public water supply improvements.

SUPPORTING LITERATURE

Berman, D. and Hoff, J. C., "Inactivation of Simian Rotavirus SA11 by Chlorine, Chlorine Dioxide, and Monochloramine," Applied and Environmental Microbiology, 48, 2, August 1984.

Collier, M. K., and Macdonald, P., "Giardiasis in Edmonton," Edmonton Local Board of Health, July, 1983.

Geldreich, E. E. , et al., "The Bacteriological Aspects of Stormwater Pollution," JWPCF, 40, 11, Nov. 1968.

Griffith, J. D., and Williams, R. G., "Application of Jar Test Results at Phoenix, Arizona," JAWWA, 64, 12, Dec. 1972.

Hoff, J. C., "Inactivation of Microbial Agents by Chemical Disinfectants," EPA Report under review, 1986.

Hudson, H. E., Jr., Water Clarification Processes-Practical Design and Evaluation, Van Nostrand-Reinhold, 1981.

Hudson, H. E., Jr., and Wagner, E. G., "Conduct and Use of Jar Tests," AWWA Seminar Proceedings on Upgrading Water Treatment Plants to Improve Water Quality, 1980.

Kreissl, J. F., Robeck, G. G., and Sommerville, G. A., "Use of Pilot Filters to Predict Optimum Chemical Feeds," JAWWA, 60, 3, March 1968.

Logsdon, G. S., "Pilot Plant Studies - From Study Planning to Project Implementation," AWWA Seminar Proceedings on Design of Pilot Plant Studies, 1982.

Logsdon, G. S., et al., "Evaluating Sedimentation and Various Filter Media for Removal of Giardia Cysts, JAWWA, 77, 2, Feb. 1985.

MacNeill, J. S., Jr., and MacNeill, Arthur, "Feasibility Study of Alternative Technology for Small Community Water Supply," U.S. EPA Project Report. Cincinnati, Ohio 1985.

"Manual for Evaluating Public Drinking Water Supplies," U.S. Public Health Service, Bureau of Water Hygiene, Cincinnati, Ohio 1969.

Murdoch, D., and Walker, G. S., "Water Treatment and Quality Control," Edmonton Water and Sanitation, Feb. 1986.

Neuman, E. W., "Optimizing Coagulation with Pilot Filters and Zeta Potential," JAWWA, 73, 9, Sept. 1981.

O'Connell, R. T. "Suspended Solids Removal," in Water Treatment Plant Design, R. L. Sanks, Editor, Ann Arbor Science, 1978.

"Recommended Standards for Water Works, 1982 Edition," Health Education Service, Albany. N.Y. 12224.

Singley, J. E., "Coagulation Control Using Jar Tests," AWWA Seminar Proceedings - Coagulation and Filtration: Back to the Basics, 1981.

Wagner, E. G., and Hudson, H. E., Jr., "Low-Dosage, High-Rate Direct Filtration," JAWWA, 74, 5, May 1982.

A Public Opinion Survey
# of Attitudes and Concerns
about Edmonton's Drinking Water

a task study for

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

by

MTK Research Associates Ltd.

Edmonton

June 1986

During the month of June, 1986, MTK Research Associates Ltd. performed a telephone survey, using numbers selected at random for all Edmonton exchanges, to assess the attitudes and concerns of Edmontonians about their drinking water. The survey was conducted by experienced interviewers using the questionaire attached. A total of 400 interviews were completed.

The choice of answers was only read to the respondent in those cases designated "R.A." on the questionaire. The questionaire was developed by Dr. Hrudey with reference to a survey conducted for the City in 1985 and in consultation with Edmonton Water and Sanitation, the Edmonton Board of Health, Alberta Environment and MTK Research Associates Ltd.

Answers to the questions are charted in the following section. In some cases, the responses to a given question only correspond to a smaller group who responded in a specified manner to an earlier question. These cases are noted directly on the charts.

1. Do you usually drink Edmonton tap water?
A. yes B. no C. Not during Spring
go to 3

---

2. How do you usually drink it?
R.A. A. as is B. only in cold drinks C. only in hot drinks D. all of these

---

3. How concerned are you about the quality of Edmonton water?
R.A. A. very concerned B. quite concerned C. not too concerned D. not at all concerned
go to 5

---

4. What is it about the water that concerns you?
A. its smell B. its taste C. its safety D. all of these E. other F. don't know

---

5. How would you describe its taste or odor at times when it is noticeable?
R.A. A. medicinal B. gasoline or oil C. bleach (chlorine) D. swampy
E. musty F. all of these G. other__________ H. no taste or odor
go to 8

---

6. When do you experience this problem?
R.A. A. only in Spring B. all or most of the year C. other__________

---

7. How frequently do you experience this noticeable taste or odor?
R.A. A. less than once a month B. once a month or more C. continuously

---

8. Do you have other water quality problems?
A. colour B. cloudy C. hardness D.other__________ E. No F.don't know

---

9. Do you think the water contains substances harmful to your health, or is it just a nuisance?
A. harmful substances B.nuisance C. both harmful and a nuisance
D. other__________ E. don't know F. neither
go to 11

---

10. Have you taken any action about the problem?
A. complained to the authorities B. installed a purifier C. used bottled water
D. do not drink water in spring E. other__________ F. No

---

11. Do you have a filter on your tap?
A. yes B. no

---

12. How often do you buy bottled or distilled water for normal drinking purposes?
R.A. A. at least once a week B. less than once a week C. never
go to 15

---

13. Have you been buying or using more of it this spring?
A. yes B. no

---

14. Do you usually buy more bottled or distilled water during the Spring?
A. yes B. no C. don't know

15. How much would you be willing to pay extra, per month, on your water bill to solve your concerns with the water?
R.A. A. nothing B. up to $2.00 C. up to $4.00 D. up to $8.00 E. up to $15.00 F. not sure
Don't Read: G. Respondent believes taxes should cover costs

---

16. How did the drinking water quality this spring compare with spring time last year?
A. better B. worse C. about the same D. don't know

---

17. The Federal and Provincial governments have quality control standards which Alberta water utilities must conform to. Do you believe these objectives are adequate?
A. yes, adequate B. no, not adequate C. don't know

---

18. Do you believe that Edmonton follows these standards adequately?
A. yes B. no C. don't know

---

19. Do you believe that Edmonton's water is better, about the same, or worse than the water in other Canadian cities such as Calgary or Toronto?
A. better B. same C. worse D. don't know

---

20. How good a job do you believe the City Water and Sanitation Department is doing at treating Edmonton's drinking water?
R.A. A. excellent B. good C. fair D. poor E. don't know

---

21. Do you believe that you have ever become sick from drinking Edmonton tap water?
A. yes B. no C. don't know
go to 24 *go to 24*

---

22. What was the nature of that illness?

---

23. What time of year was it when you became ill - was it Spring, Summer, Fall, or Winter?
A. Spring B. Summer C. Fall D. Winter E. don't know

---

24. Do you believe it is possible to make Edmonton's drinking water safer than it is now?
A. yes B. no C. don't know

---

25. Do you believe that you have been given sufficient information on regular testing of our water?
A. yes B. no C. don't know

Some questions for statistical purposes:
26. How long have you lived in Edmonton?
A. less than 1 year B. 1-2y C. 3-5y D.5-10y E.more than 10y

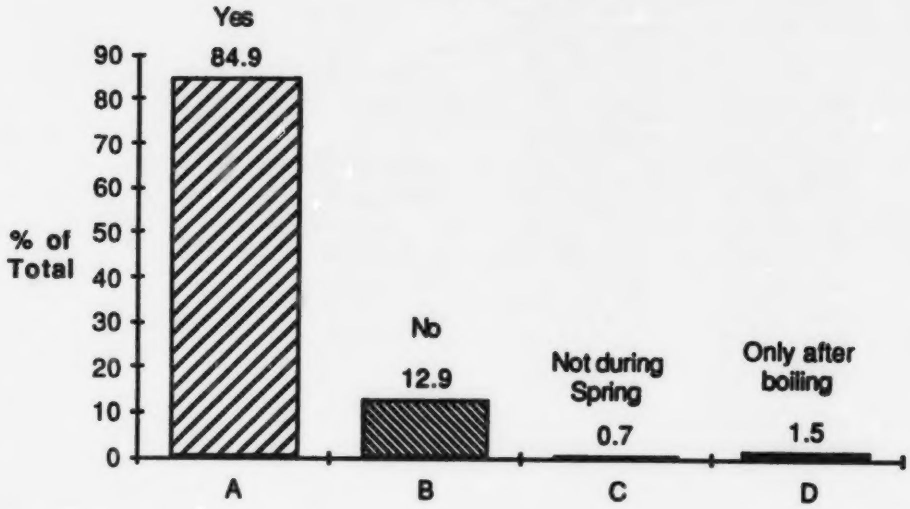
---

27. Which of these categories best describes your age?
A.18-24 B.25-34 C.35-54 D.55-64 F.64+

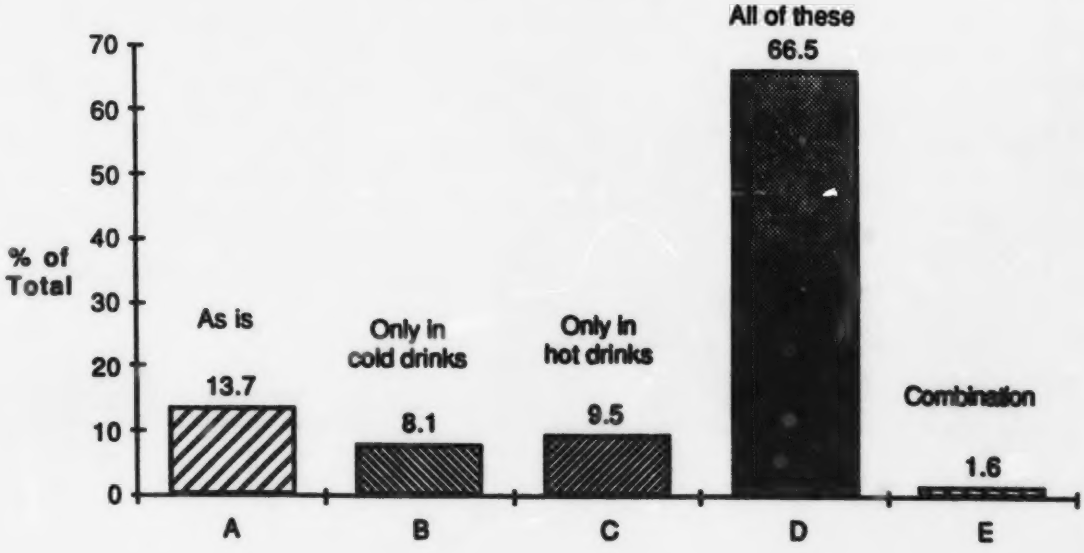
---

29. A. male B. female

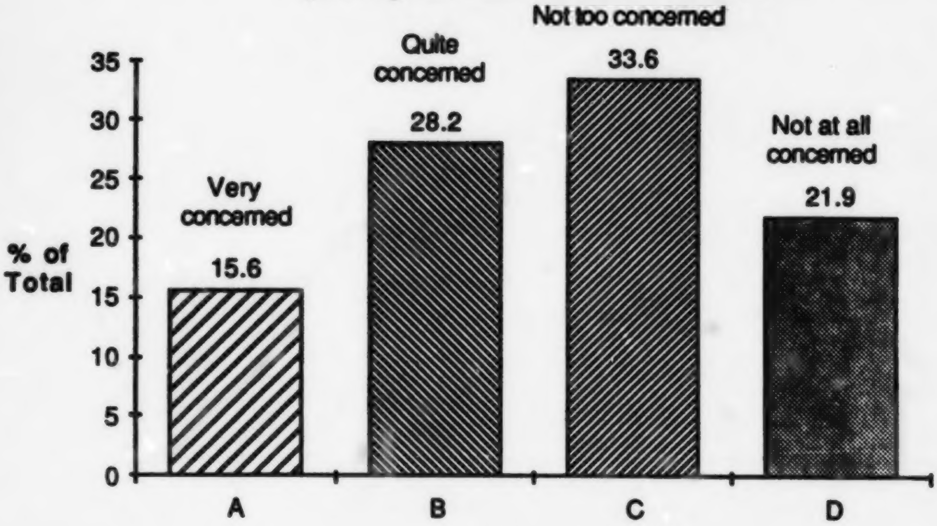
Q1. Do you usually drink Edmonton tap water?



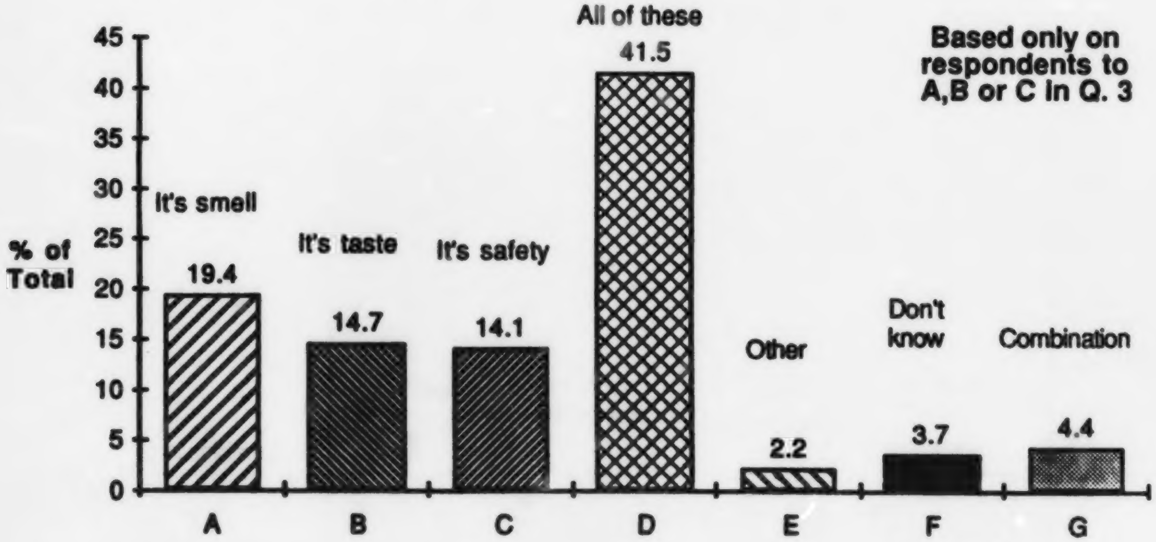
Q2. How do you usually drink it?



Q3. How concerned are you about the quality of Edmonton water?

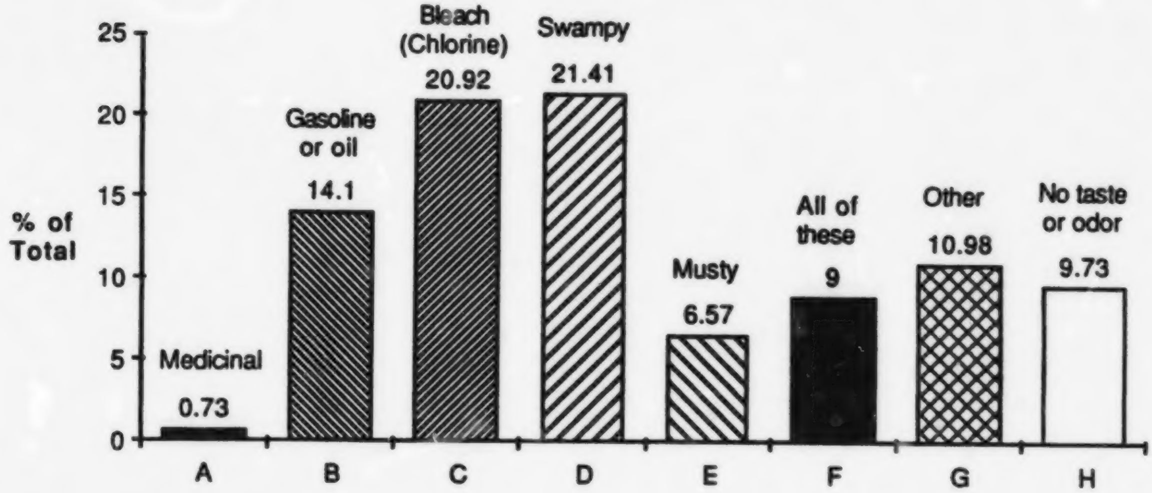


Q4. What is it about the water that concerns you?

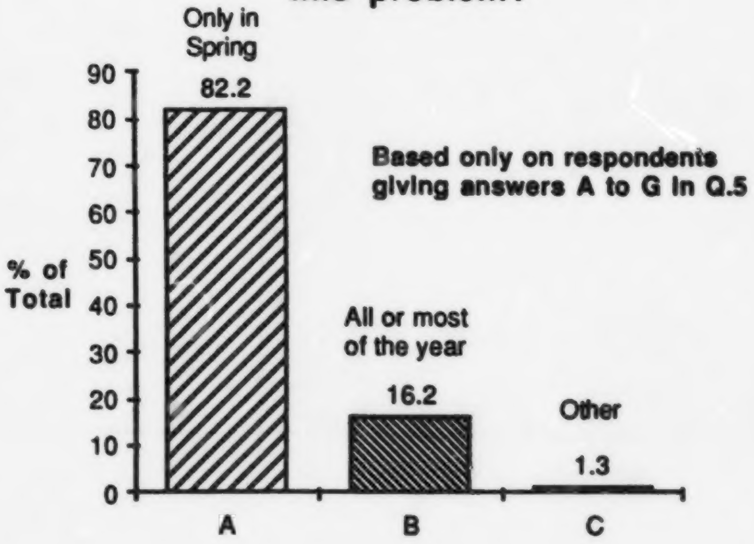


Based only on respondents to A,B or C in Q. 3

Q5. How would you describe its taste or odor at times when it is noticeable?

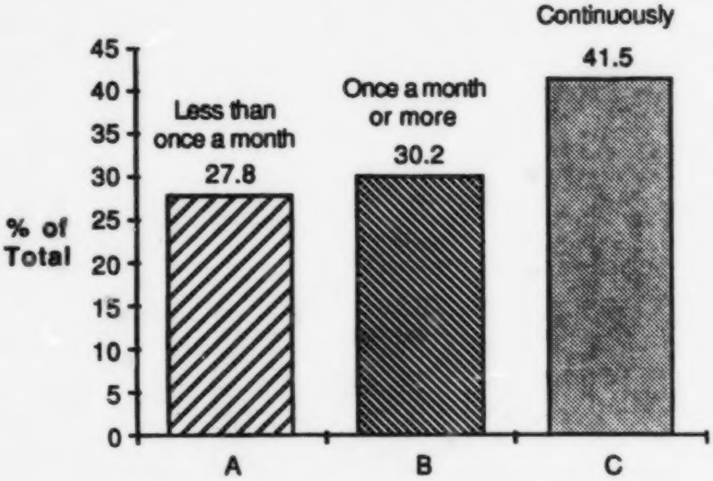


Q6. When do you experience this problem?



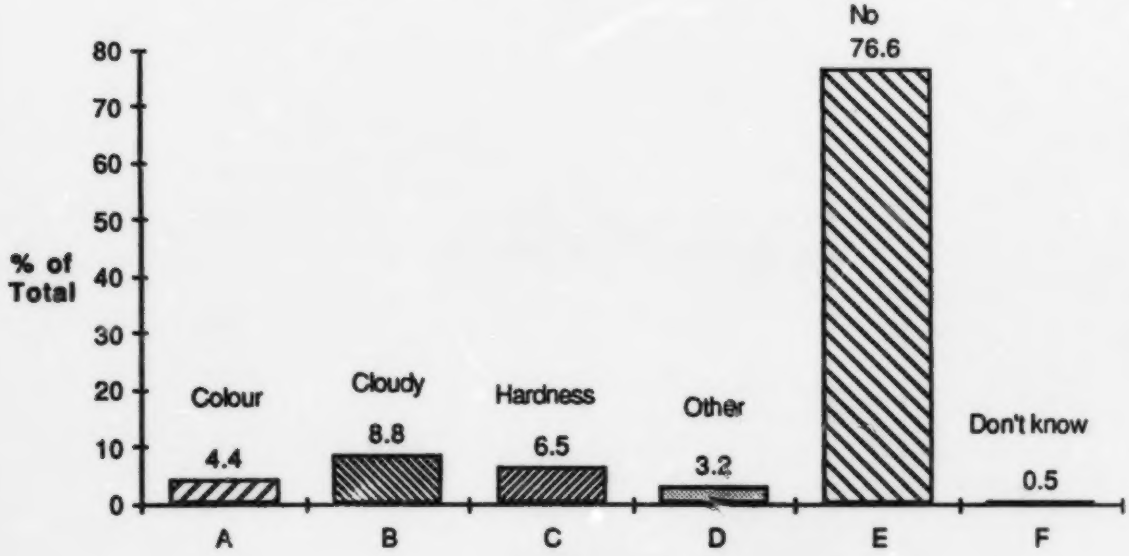
Based only on respondents giving answers A to G in Q.5

Q7. How frequently do you experience this noticeable taste or odor?

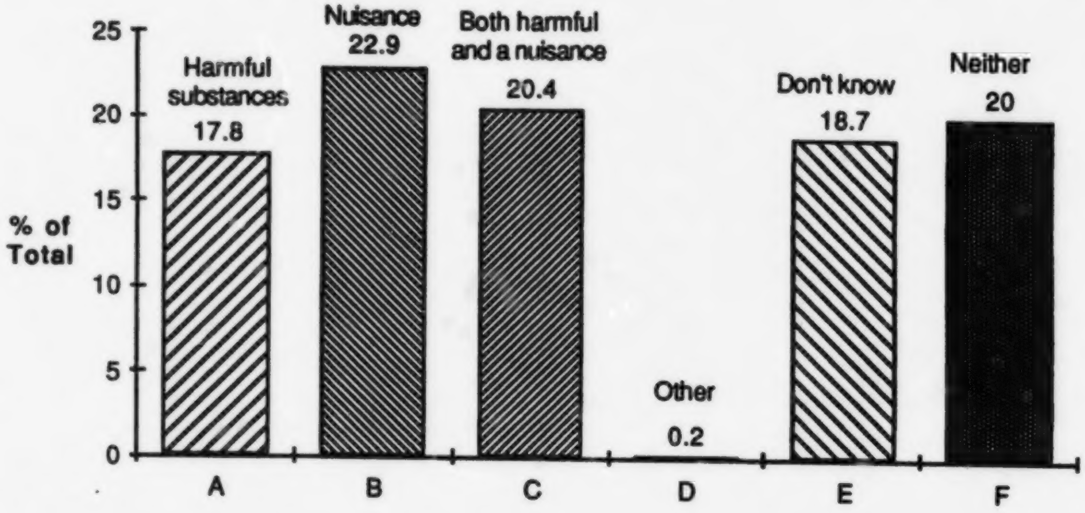


Based only on respondents giving answers A to G in Q.5

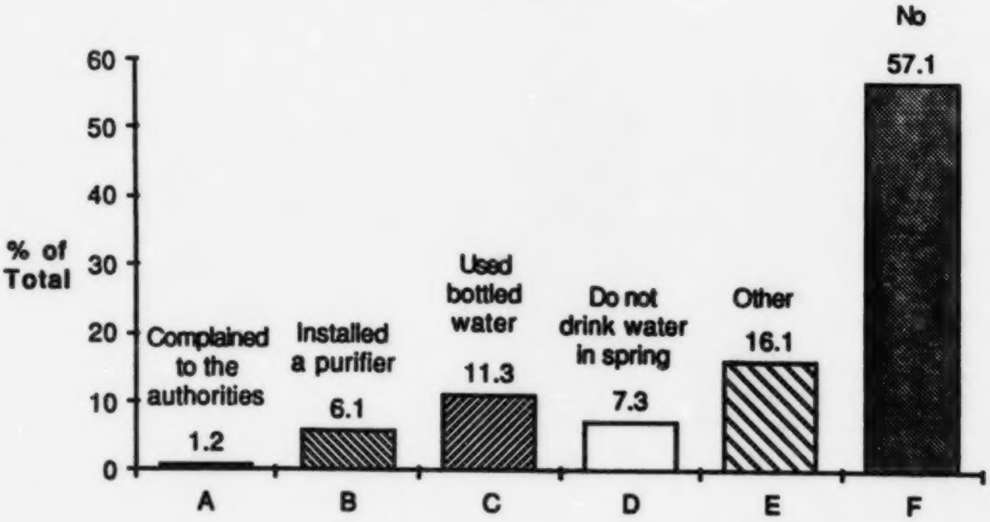
Q8. Do you have other water quality problems?



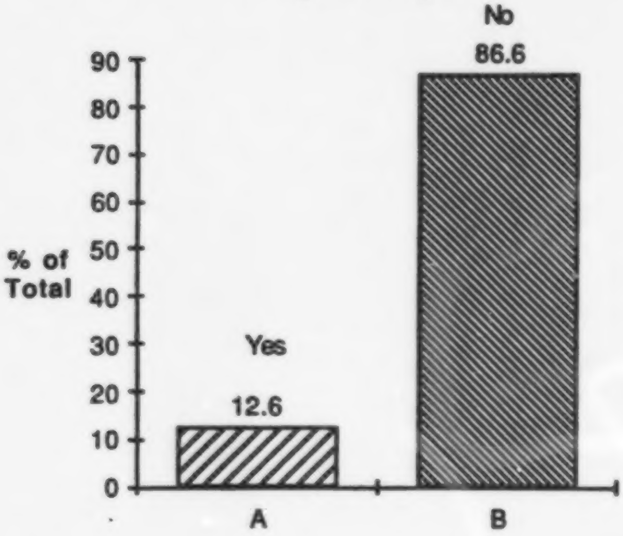
Q9. Do you think the water contains substances harmful to your health, or is it just a nuisance?



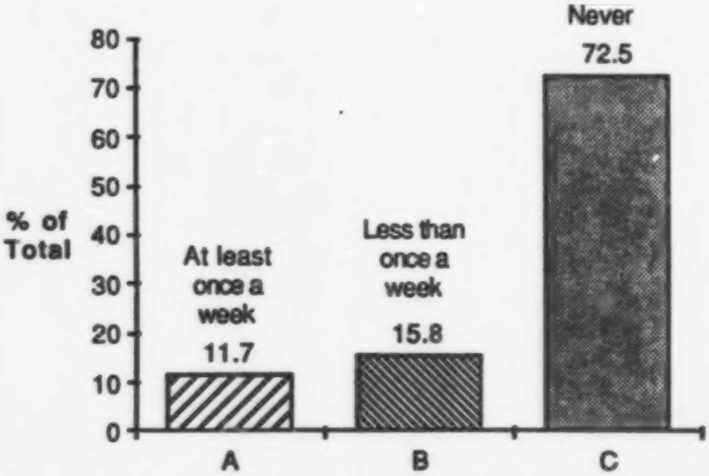
Q10. Have you taken any action about the problem?



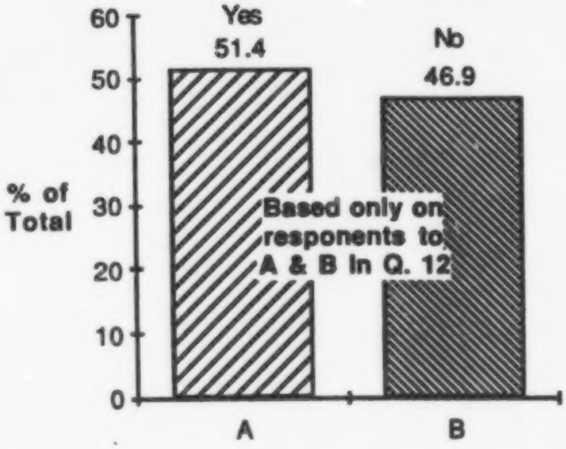
Q11. Do you have a filter on your tap?



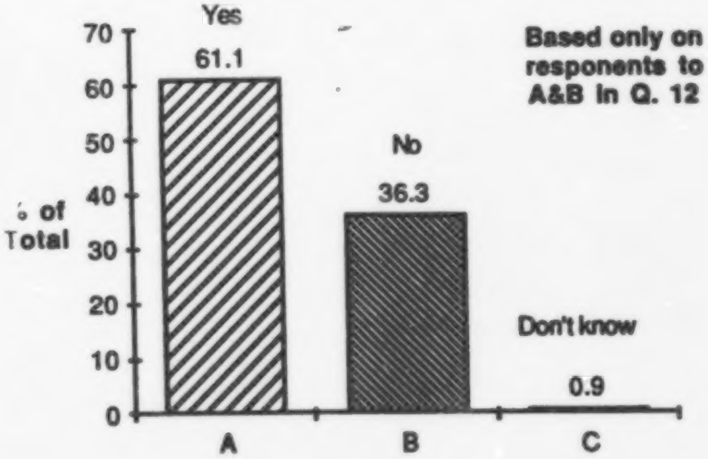
Q12. How often do you buy bottled or distilled water for drinking purposes?



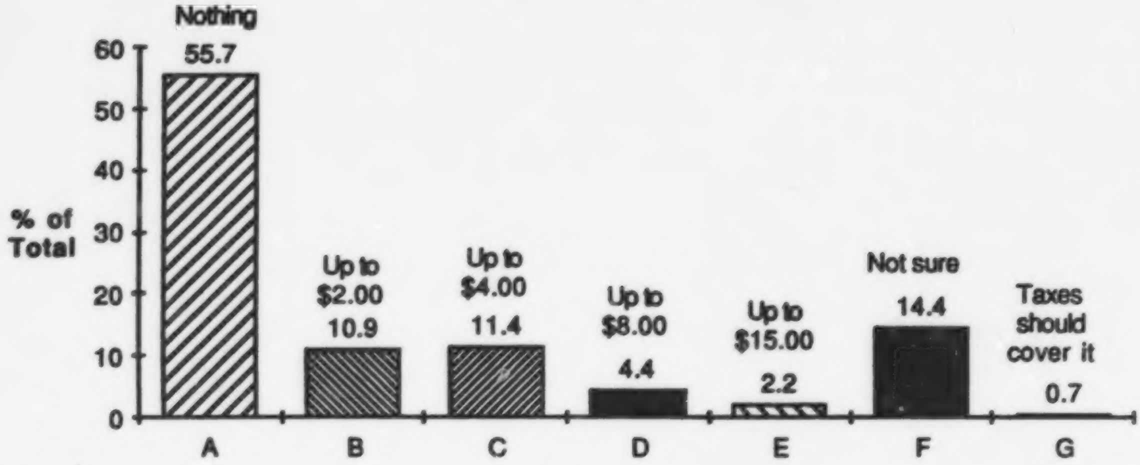
Q13. Have you been buying or using more of it this spring?



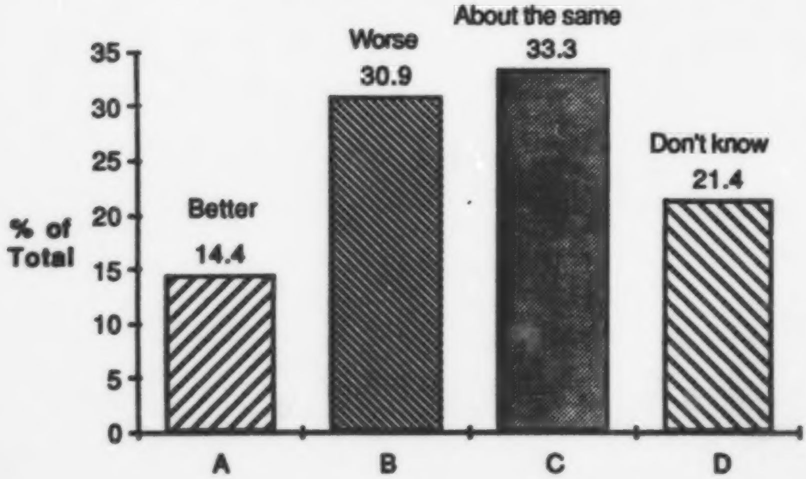
Q14. Do you usually buy more bottled or distilled water during the spring?



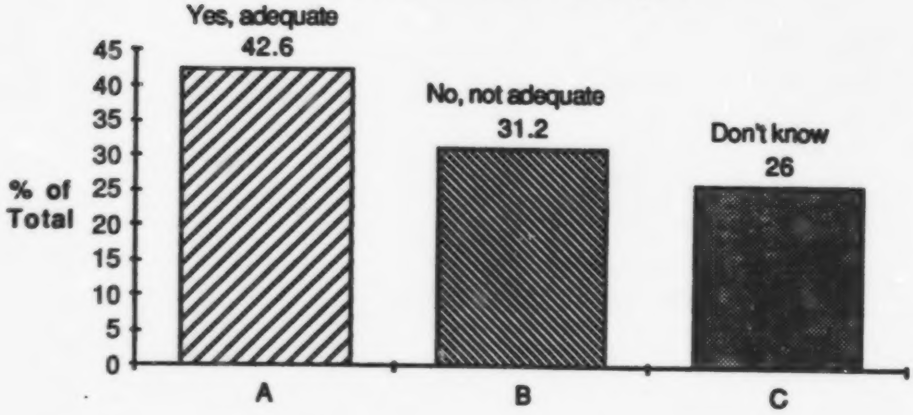
Q15. How much would you be willing to pay extra, per month, on your water bill to solve your concerns with the water?



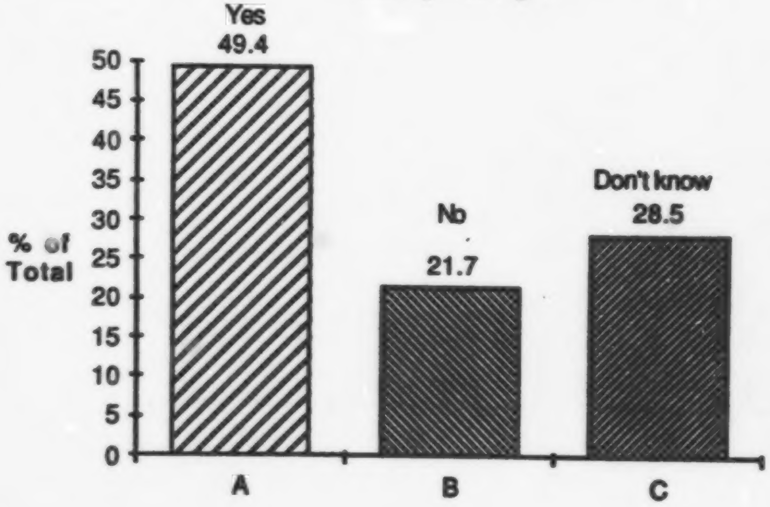
Q16. How did the drinking water quality this spring compare with spring time last year?



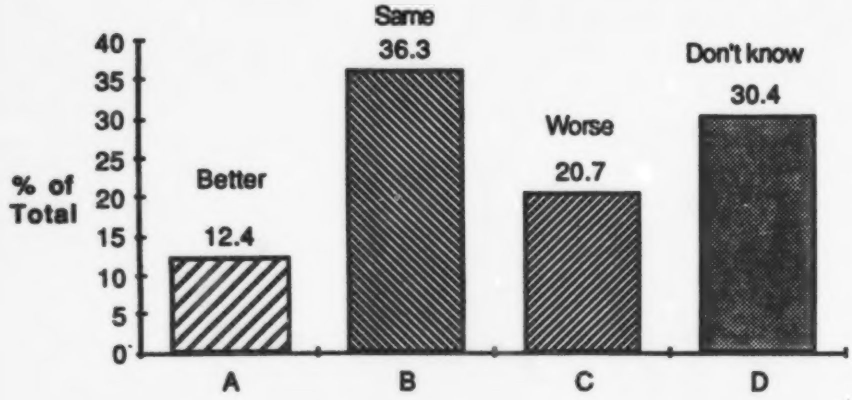
Q17. The Federal and Provincial governments have quality control standards which Alberta water utilities must conform to. Do you believe these objectives are adequate?



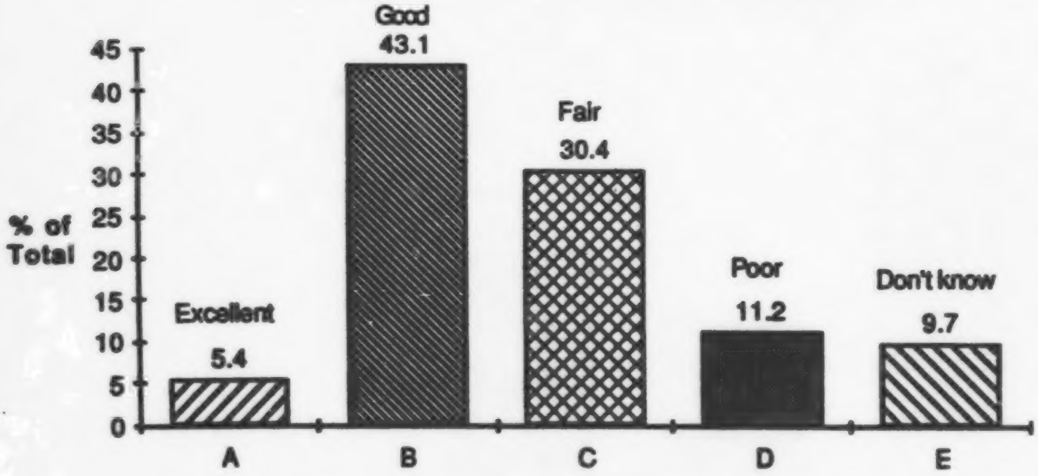
Q18. Do you believe that Edmonton follows these standards adequately?



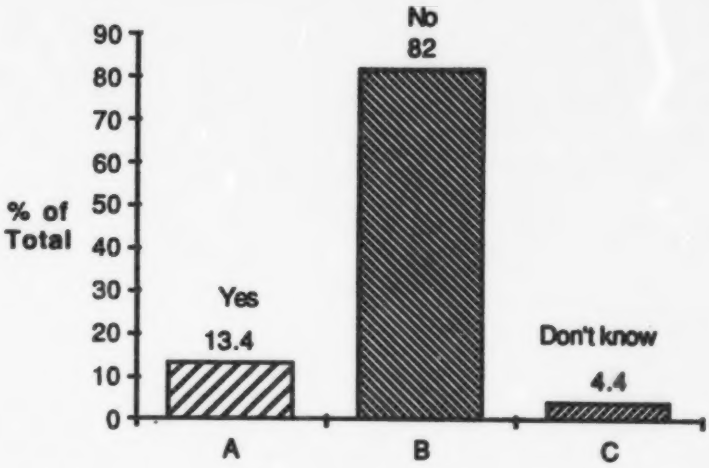
Q19. Do you believe that Edmonton's water is better, about the same, or worse than the water in other Canadian cities such as Calgary or Toronto?



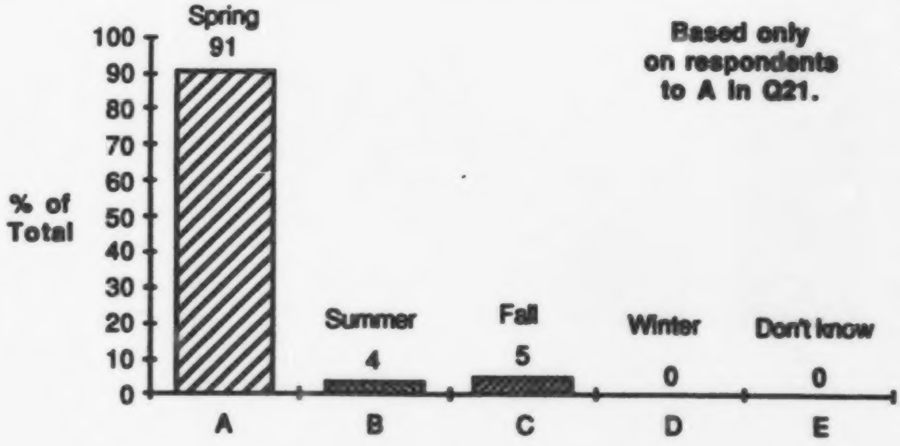
Q20. How good a job do you believe the City Water and Sanitation Department is doing at treating Edmonton's drinking water?



Q21. Do you believe that you have ever become sick from drinking Edmonton tap water?

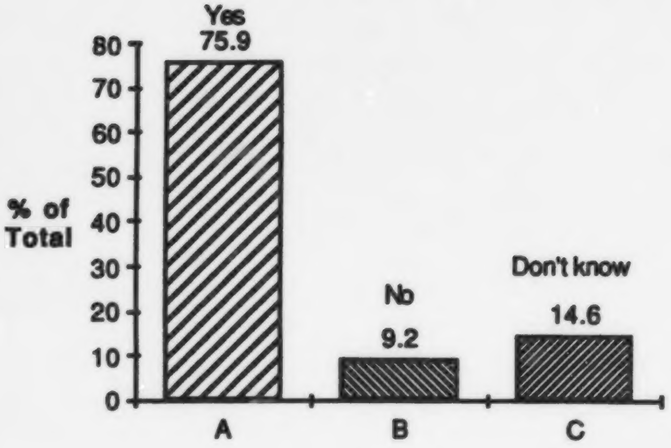


Q23. What time of year was it when you became ill - Spring, Summer, Fall, or Winter?

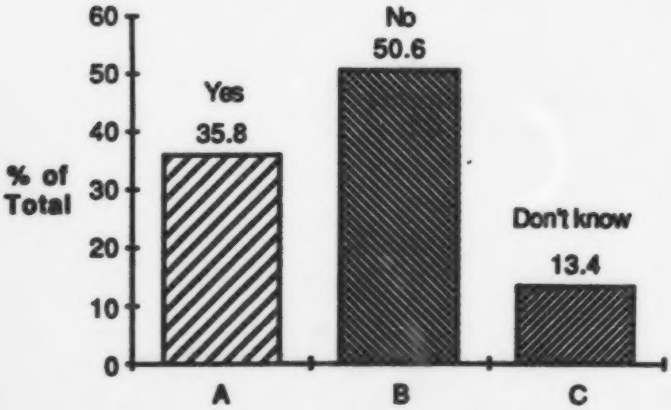


Based only on respondents to A in Q21.

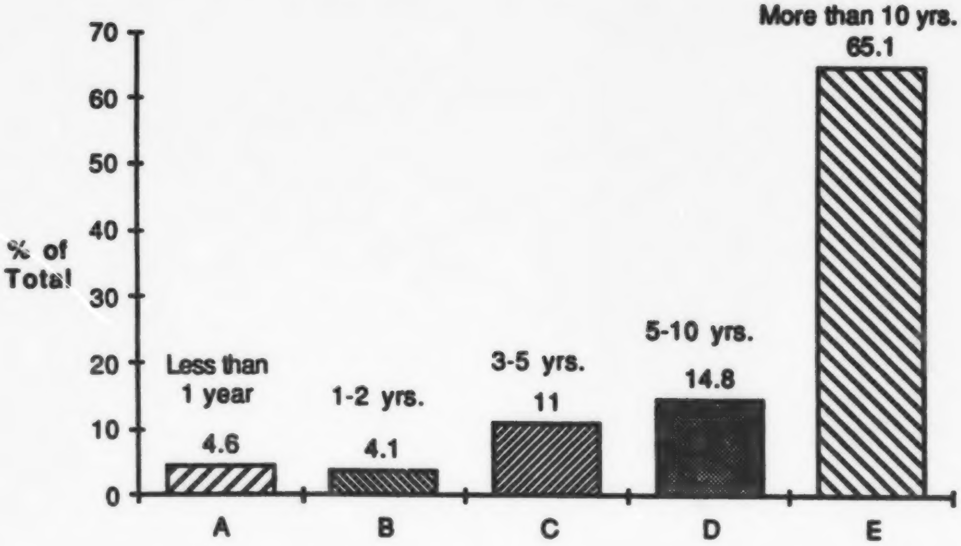
Q24. Do you believe it is possible to make Edmonton's drinking water safer than it is now?



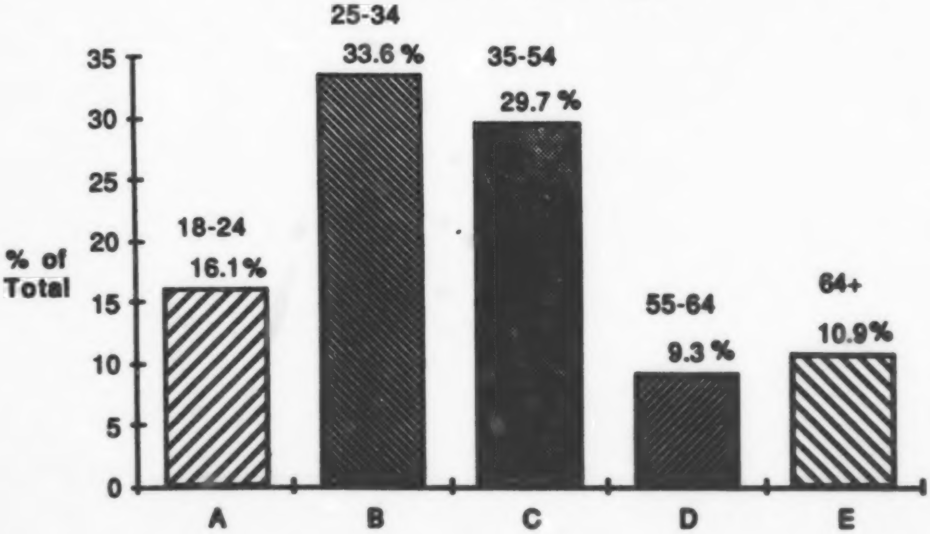
Q25. Do you believe that you have been given sufficient information on regular testing of our water?



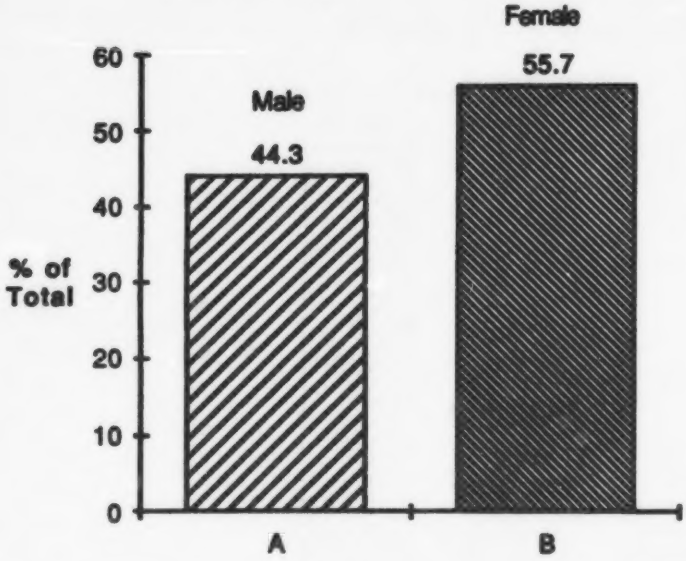
Q26. How long have you lived in Edmonton?



Q27. Which of these categories best describes your age?



Q29. Are you male or female?



EVALUATION OF VIRAL INDICATORS AND PATHOGENS IN WATER FROM THE EDMONTON AREA

Report presented to

Steve Hrudey & Associates Ltd
and the
City of Edmonton

by

Pierre Payment, PhD

Centre de Recherche en Virologie
Institut Armand-Frappier
C.P.100, Succ. L.D.R.
Laval, Quebec, H7N 4Z3

March 1986.

SUMMARY

The following objectives were set to us for this report:

1. Assess or propose virological analysis of water in the Edmonton metropolitan area. (Task 3.2.3. item 1 and 4)

2. Assess current water treatment practices in relation to the current water source and the possible health risks due to viral contamination. (Task 3.2.3. item 2 and 3 and 5)

The conclusions of the present report on these objectives are:

1. Weekly virological analysis of the current water source and of the finished water from all water treatment plants should be performed. The procedures to be used should be in accordance with current EPA methods for virus concentration and detection and in close collaboration with an environmental virologist.

2. A change of the water intake location of the Rossdale plant, if it results in a clear improvement of the bacteriological quality of the source water , will reduce the risk of viruses and other pethogens penetrating the water distribution system. Under normal circumstances, the current water treatment practices should be sufficient to prevent any epidemic of viral disease. However, any failure or reduced efficiency in the treatment processes could result in a higher incidence of gastrointestinal diseases or epidemic hepatitis in the population due to the breakthrough of pathogenic microorganisms including viruses. The risk of such an event occuring is certainly more elevated at the Rossdale plant due to the poorer source water quality and plant condition.

3. Current disinfection methods using chlorine dioxide plus chloramination should be sufficient to control pathogenic microorganisms including viruses, provided that the raw water used is relatively free of contamination and that flocculation and filtration treatments are optimal. However, the use of ozone for the control of taste and odor and the use of chlorine for disinfection could be considered as safe alternatives.

INTRODUCTION

This report is the result of a two-day site visit in Edmonton on February 26 and 27 1986 and of a review of the current literature on the subject of viruses in drinking water. The main objective of this report was to assess the efficiency of removal of viruses by the two water filtration plants of the city of Edmonton, assess the health risk and provide recommendations for future actions if required.

BACKGROUND INFORMATION OBTAINED DURING THE SITE VISIT

Water filtration plants

The Rossdale filtration plant was built in 1947 and now distribute water that is treated by flocculation with alum and polymers, settling, filtration and disinfection with chlorine dioxide and chloramination. Water is produced "on-demand" and this practice appears to be the source of treatment problems by stretching the plant near or over its treatment capacity. Source water is obtained from the river by a shore-located intake which is occasionally subjected to heavy chemical and storm-sewer pollution from the city of Edmonton itself. Some heated water from a nearby utility is used to reduce freezing problems in some older parts of the distribution system. The only mean of preventing improperly treated water from entering the distribution system is drainage of the plant. The only alternative is a "boiling water notice to all consumers" if a major treatment failure is encountered (i.e. presence of turbidity higher than normal or failure to maintain disinfectant level even for a short period). The reservoir that is being built will act as a buffer and give plant operators some precious hours to warn th public in case of failure.

The E.L.Smith filtration plant was opened in 1976 and incorporates most of the advanced methods of control of the water purification processes. The complete treatment includes flocculation by alum and polymers, settling, filtration and chlorine dioxide /chloramine disinfection. Source water is obtained from the central part of the river. Finished water is kept in reservoirs sufficient for about a half-day demand and in case of treatment failure water can be diverted directly to the river before entering the distribution system.

Water quality

Water is abstracted from the North Saskatchewan River which originates from the Saskatchewan Glacier in the Rocky Mountains. The river is also fed by three rivers (Brazeau, Clearwater and Nordegg) flowing through muskeg and bushland areas. Odor, taste, high organic load and turbidity result from both spring snow meltdown and heavy rains.

Water quality parameters of importance to the microbiological and virological quality of the treated water have been selected from the "Water Treatment Plants, 1984 Annual Report, City of Edmonton" and are presented in table 1.

TABLE 1. Water quality parameters values for 1984 at the filtration plants.

	ROSSDALE			E.L.SMITH	
	Raw	Treated 1-2	Treated 3	Raw	Treated
Chlor.res. (mg/1)	n.a.	- (-).26	- (-).42	n.a.	- (-).20
Chlor.free "	n.a.	- (-).37	- (-).10	n.a.	- (-).05
Chlor.Total "	n.a.	1.3(1.9)2.2	1.8(1.9)2.1	n.a.	1.6(1.8)2.1
Color (TCU)	2 (10) 57	1(1)5	1(1)3	1(9)47	1(1)9
pH	7.7(8.2)8.6	7.5(8.3)8.6	7.9(8.3)8.5	7.8(8.2)8.5	8.0(8.4)8.6
Turbidity (NTU)	1(10)148	.1(.4)1.0	.2(.4).8	1(10)178	.1(.3)1.3
Fecal coliforms*	8(140)1600	-	-	-(>5)>320	-
Total coliforms	-(>1100)>24000	-(-)5	-	-(72)5400	-(-).16

*Fecal and total coliforms expressed as CFU/100ml
n.a.: not applicable (-) = negative

Bacteriologically , raw water quality is markedly different at the Smith Plant and at the Rossdale Plant due to sewage outlets discharging their content in the river near the Rossdale Plant. Fecal coliforms densities are less than 200/100ml at the E.L.Smith plant while they can attain 4000/100ml at the Rossdale plant. Storm sewers situated near the plant affect the source water quality during the spring run-off: the water is then heavily contaminated with dirt from the streets. Cross contamination between sanitary sewers and storm sewers (a few percent) was also reported to us (personal communication, Steve Hrudey). Preliminary data for February and March 1986 indicates that the Groat sewer wastewater contained mainly material of mixed origins (human/ animal) as Fecal Coliform/ Fecal Streptococci ratios were usually between 0.5 and 4. Other sewers and the source water have not been tested.

Fecal coliform bacteria numbers are in a range that requires a complete treatment of the water to eliminate the risk of waterborne outbreaks. As shown by the giardiasis outbreak that occurred a few years ago the quality of treatment at the time may not have been not sufficient to eliminate the more resistant pathogens.

Turbidity can be very elevated at both plant during the spring ice melt-down. This difficult situation stresses the Rossdale Plant to its operational limits and, as mentioned earlier, there is no possibility of diverting improperly treated water back to the river. High turbidity could be the cause of a breakthrough of pathogenic microorganisms in the distribution system and would be one of the major causes for concern.

Chlorine dioxide is used at both plants. The continuous use of chlorine dioxide was adopted in 1983 as were other modifications of treatment as the result of an important epidemic of *Giardia lamblia* . At both plants, raw and finished waters are over pH 8.0, and over pH 9.5 during most of the treatment processes. The virucidal activity of chlorine dioxide would be maintained at elevated pH values as it is less susceptible to pH variations than chlorine. Free residual chlorine is not maintained after treatment: only Total Chlorine is present probably as chloramine. A residual cannot be maintained in high demand period if the treated water is to meet the chlorine dioxide plus chlorite maximum level of 0.5mg/l set by the province. The microbiocidal activity of chloramines could be adequate for bacteria but their virucidal activity is slow and longer contact times required (Hoff and Geldreich, 1981). Furthermore, some bacteria may be more resistant than other to chloramine disinfection and their efficiency related to the mode of application and pH (Wolfe et al,1985).

Finished water meets current standards of water quality in Canada. A total combined chlorine concentration of about 2.0 mg/litre is maintained in the treated water and the presence of the chloramines may be sufficient to insure some degree of protection against contamination of the distribution system.

Virological analysis has been performed on a preliminary basis on a few samples of river water and sewage submitted by the Ministry of Environment to the Provincial Public Health Laboratory for virus isolation. Viruses were detected in two of the samples but the results are only qualitative and are of little value for the present report.

GENERAL COMMENTS ON WATER TREATMENT AND QUALITY

Microbiological quality

The microbial contamination of the source water can originate from two sources: an animal source (wild or domestic) and a human source (untreated domestic sewage discharges). The viruses from animal sources will most probably not cause any human health problem as the transmission of enteric viruses from animal to man is a very rare occurrence. The major problems associated with animal fecal material will be due to bacterial pathogens (Salmonella , Shigella , Campylobacter , etc...) and parasites (Giardia lamblia , amoebae, etc...)

The contamination by fecal material of human origin, apart from being the source of human bacterial and parasitic pathogens, will also result in the presence of human enteric viruses including those responsible for gastroenteritis, poliomyelitis and hepatitis. Viruses can survive water treatments including disinfection and their isolation in finished water is in correlation only with their presence in source water (Payment et al,1985a).

Due to the relatively high levels of contamination by fecal coliform bacteria at the Rossdale plant and to the undefined origin of these fecal bacteria, the presence of viruses in the drinking water must be considered possible until proper analysis have been performed. The bacterial content of the Groat sewer near the Rossdale plant indicates that the pollution is of mixed origin but with some human feces content because the FC/FS ratios measured in February and March were often between 1 and 4.

The information available to us on the human population upstream the Smith plant indicates that the human fecal pollution should not be of major concern at that plant. This correlates with the low fecal coliforms content. Further analysis might not be necessary at the Rossdale plant if the inlet is moved to the Smith plant. Weekly analysis should be considered if the inlet is not changed or simply moved further to the center or the North bank of the river. The lower level of fecal coliform bacteria at the E.L.Smith plant indicates a lower risk of human viral pollution. The facts that the human population upstream is small and that the nearest town (30km upstream, Devon) treats its wastewater, suggest that the risk of a viral pollution is low. However, ecause viruses are more resistant than fecal coliform bacteria in water, the level of the viral pollution could be more elevated than expected. In the absence of data on the fecal pollution of the North Saskatchewan River, this hypothesis cannot be assessed and adequate monitoring of the river water at the inlet of the plants should be instigated.

Treatment procedures

The current treatment procedures at both plants have theoretical virus removal capabilities of more than 12 logs (Leong, 1983; Olson and Nagy, 1983). In practice however, this level is probably never attained because of the many variables to be controlled. Coagulation, flocculation and sedimentation when properly performed should remove approximately 99% of the virus present. Filtration when performed in the best operating conditions will eliminate less than 90% of the viruses: it only removes viruses associated with large particulates and very little of the free or small particulate-associated viruses. Disinfection by chlorination and/ozonation will generally remove about 99.9% of viruses but, because all viruses are not eliminated with the same efficiency, some may resist this treatment (Bitton et al, 1983; Payment et al, 1985a et 1985b). Chloramine disinfection may not be best choice for virus inactivation because it is slower than disinfection by chlorine. Our own data in the Montreal area has not shown major differences between plants using chlorine or chlorine dioxide: viruses were found in the treated water at all plants.

VIRUSES IN DRINKING WATER: AN EVALUATION OF THE PROBLEM IN RELATION TO THE CITY OF EDMONTON WATER SUPPLY

Virus sources and inactivation

All human enteric viruses can be excreted in the feces of infected individuals and more than 100 virus types have been described. Without describing all of these viruses, we may mention that viruses are responsible for a large number of gastroenteritis symptoms, poliomyelitis, epidemic viral hepatitis, aseptic meningitis. The most frequent waterborne diseases caused by viruses in the recent years are the epidemic hepatitis (hepatitis A virus) and viral gastroenteritis (Norwalk's agent, Snow Mountain agent, and rotaviruses) . On any given day, even in the absence of evident clinical symptoms, up to 1 out of 10 individuals can be excreting in their feces billions of infectious particles of any of these viruses: sanitary sewers will thus act as the vehicle of the virus-containing fecal material to surface waters.

Viruses are usually more resistant to natural inactivation than bacteria , but less than cysts of parasites like Giardia lamblia . Viruses can survive for weeks and even months in water and sediments but, because they are obligate parasites (they must infect their host, man in this case, to multiply) they cannot multiply in the aquatic environment. The lower the water temperature the longer will virus survive. On the contrary, bacteria will usually survive a few hours to a few days in water, but occasionally when water temperature increases they can multiply using available nutrients in water.

Viruses are more resistant than bacteria and some parasites to water treatment processes , including disinfection. Some pathogenic viruses have been shown to resist for hours in the presence of free residual chlorine: at a level of 1 mg/L free residual chlorine it may take several hours to inactivate Coxsackieviruses in river water by 99%. Wastewater treatment facilities will reduce the indigenous viral population of sewage by 90% in the absence of disinfection and 98-99% with disinfection. Other water treatment processes will be efficient in removing viruses only if properly performed. As mentioned earlier, the practice of using alum flocculation at pH values above 7.0 may be questioned. This was already mentioned by Dr Lippy of the US EPA in his review of treatments practices at the water treatment plants of Edmonton following the giardiasis epidemic. If this practice does not result in the penetration of indicator microorganisms through the flocculation process it may be continued. I would not recommend it if higher turbidity is encountered on the filters or if microorganisms are found in greater numbers after filtration.

Virus density in source and treated water

Based on information available on the bacteriological quality of source water at the two filtration plants and from data obtained at similar plants in the Montreal area (Payment et al, 1985a), I would estimate that given the fecal coliform densities in the water from the North Saskatchewan River it may contain 10 to 100 cultivable viruses /liter at the Rossdale plant and 1 to 10 viruses /liter at the E.L.Smith plant. From these values and assuming an efficacy of treatment of 99.99% at the Rossdale plant and 99.999% at the E.L.Smith plant. Viruses in the distributed drinking water could attain levels of 1 to 10 viruses/ 1000L for the applied treatments.

Health risks

While such number of viruses do not appear to be the cause of waterborne epidemics where they have been detected (Bitton et al,1983; Payment, 1981; Payment et al, 1985) they can be the source of sporadic cases of disease or subclinical infections which will not be considered as waterborne.

Viruses have very low minimal infective dose: a few infectious particles are sufficient to initiate infection. For bacteria, the minimal infective dose is usually several order of magnitude higher than for viruses. The health risk associated with the consumption of water containing viruses at such levels has been estimated by Dr Gerba (1984). His estimations are based on two infection rates (percent of exposed individuals that will become infected): a 1% infection rate as been described for polioviruses while a 30% infection rate has been reported for the Norwalk virus (Table 2).

Table 2. Virus concentration and expected infection incidence

	Number of infections/year if infection rate is	
Virus/L	1%	30%
1/ 10L	139 000	4 100 000
1/ 100L	13 900	416 000
1/ 1000L	1 390	41 600

Calculations based on a community of 190 000 individuals and two liters ingested per day.

There has not been any epidemiological study on the health effects of these low concentrations of viruses in drinking water but some environmental virologists have suggested maximum acceptable limits. The World Health Organization (1984) has included recommendations for the virological quality of drinking water. but considers that water has been adequately treated when all the following conditions are met:

- a turbidity of 1 NTU or less before disinfection
- disinfection with at least 0.5 mg/litre of free residual chlorine at a pH below 8.0

These recommendations also state that:

> "When virological facilities can be provided, it is desirable to examine the raw water sources and the finished drinking water for the presence of viruses"

Given this recommendation for virus-testing of drinking water, a monitoring should be instigated unless (Gerba, 1984):

- the watershed has no source of domestic wastes (including septic tanks)
- a complete treatment is applied to water containing less than 500 fecal coliforms per 100 ml

- travel time of water flow from point of sewage discharge to abstraction results in 8 to 10 logs of virus inactivation.

Two approaches for sampling frequency have been presented by Dr.Gerba. The first one states that: "Water systems which abstract their water from highly polluted sources should also be required to sample at least monthly to ensure that the treatment was adequate. At least quarterly sampling would be necessary to take into consideration seasonal changes in water quality and viruses." The second approach is similar to the coliform analysis frequency tables as a function of the population served:

Table.3 Virus monitoring frequency.

System size (persons)	# analysis per year	Cost(approx)
25-1000	4	$1,400
1000-10 000	6	$2,100
10 000-20 000	12	$4,200
20 000-30 000	26	$9,100
> 30 000	52	$18,200

Virological analysis

Methods for the virological analysis of water are available (USEPA, 1984; APHA, 1985) but due to the rapidly changing methods in this field the help of a competent environmental water virologist should be sought to plan and interpret the results. I have included in Annex 1 a protocol which should be feasible with the collaboration of the operators of the plants, the City of Edmonton and the virologists from the University of Alberta and the Provincial Public Health Laboratory.

The cost for virological analysis will depend on the cost of available manpower resources but as an estimate here are the values used in our own laboratory for a total cost of $700 for sampling and analysis of one 100 liter sample (raw) and one 1000 liter sample (finished) water at one filtration plant:

a. Material: ($125)
  - filters, tissue culture medium and flasks, disposables, chemicals
b. Personnel: (per local salary scales)
  - sampling: 100 liters..................= 0.5 hrs + travel
    1000 liters..................= 2.0 hrs + travel
  - processing of filters/reconcentration...= 2.0 hrs
  - cell culture and virus titration........= 4.5 hrs
c. Administrative costs / Indirect costs: (local)

These cost could be certainly lessened by proper agreements between the Ministry of Environment or the City of Edmonton (for sampling) and the Provincial Laboratory of Public Health (processing and virus detection).

SPECIFIC RECOMMENDATIONS

1. Establish the origin of the observed fecal pollution of the North Saskatchewan river using engineering data (sewer and septic tanks location) and still better, bacteriological analyses (total and fecal coliforms, fecal streptococci) to determine the microbial content of the source water by weekly analysis at each plant.

Rationale: If the sources of fecal pollution in the raw water treated by the filtration plants are of animal origin, viruses will not be a human health risk because animal enteric viruses are rarely transmitted to man. Other microorganisms (bacteria and parasites) will however remain a problem.

2. Move the raw water inlet of the Rossdale plant to a less contaminated location (i.e.: North bank of the river or better, upstream). Source water quality should be similar or better than observed at the E.L.Smith plant.

Rationale: because of the older nature of the plant and its perceived flaws it is less risky to treat water that is contaminated as little as possible by fecal material. While this plant can produce drinking water free of indicator bacteria and meeting current water quality standards, I doubt that in normal as well as emergency situation it can produce water that is virus-free if it treats contaminated source water.

3. Establish a program of virological testing of both source and treated water. This could be a temporary intensive program (biweekly) followed later by occasional surveys at less frequent intervals (quarterly).

Rationale: While no standard has been set for viruses in drinking water, it appears that drinking water prepared from surface water contaminated by human fecal material should be free of viruses. Because of their resistance to water treatments especially filtration and chlorination, the presence of viruses in the treated water would be indicative of inadequate water treatments. The evaluation of the viral pollution of the source water as well as of the drinking water will permit the assessment of this efficiency.

Note : If the water inlet of the Rossdale plant is moved to the E.L.Smith plant in a nearby future, testing should also be performed to assess the efficiency of both plants to treat the same water.

4. Evaluate the efficiency of the "winter mode of treatment" which uses high pH treatment of the water.

Rationale: Because flocculation is performed at pH values above 7.0 during winter, its efficiency could be reduced. It would be important to review its efficiency for the removal of turbidity as well as other physico-chemical and microbial parameters.

5. Evaluate the use of an alternative disinfectant like ozone to control odor and taste while retaining a chlorine based disinfectant with an active residual for the disinfection of the water and for protection of the distribution system.

Rationale: If the chlorine dioxide plus chlorite maximum of 0.5mg/l does not permit the obtainment of a free residual chlorine dioxide residual, I am concerned on the efficacy of the chloramines alone to control pathogenic microorganisms especially viruses. As chlorine dioxide was originally used to control odor and taste, and only later as a biocidal agent, it may be more efficient to use ozone for physico-chemical control and chlorine for disinfection thus eliminating taste/odor problems and the formation of chlorinated compounds.

SUPPORTING LITERATURE

- , (1978) Guidelines for Canadian Drinking Water Quality, Health and Welfare Canada, Publication H48-10/1978.

American Public Health Association (1985) Standard Methods for the Examination of Water and Wastewater, 16th Ed., p.946-974.

Bitton,G. et al (1983) Survey of virus isolation data from environmental samples. Report to USEPA for project 68-03-3196.

Gerba,C.P. (1984) Strategies for the control of viruses in drinking water. Report to AAAS/EPA, 87 pages.

Hoff,J.C. and E.E.Geldreich (1981) Comparison of the biocidal efficiency of alternative disinfectants. J.A.W.W.A. 73:40-44.

Leong, L.Y.C. (1983) Removal and inactivation of viruses by treatment processes for potable water and wastewater. Water Science and Technology 15:91-114.

Olson,B.H. and L.A.Nagy (1984) Microbiology of potable water. Advances in Applied Microbiology 30:73-132.

Payment, P., R.Plante et M.Trudel (1985a) Elimination of viruses and indicator bacteria at each step of treatment during preparation of drinking water at seven water filtration plants. Applied and Environmental Microbiology 49:1418-1428.

Payment,P., M.Tremblay and M.Trudel (1985b) Relative resistance of poliovirus and coxsackievirus isolates from environmental sources. Applied and Environmental Microbiology 49:981-983.

Sproul,O.J. (1983) Public health, financial and practical considerations of virological monitoring and quality limits. Water Science and Technology 15:33-41.

US Environmental Protection Agency (1984) USEPA Manual of methods for virology. Publication EPA-600/4-84-013

Wolfe,R.L., N.R.Ward and B.H.Olson (1985) Inactivation of heterotrophic bacterial populations in finished drinking water by chlorine and chloramines. Water Res. 11:1393-1403.

World Health Organization (1984) Guidelines for drinking-water quality. Vol.1 Recommendations, WHO Geneva.

ANNEX 1

Virological analysis of water (VIRADEL Method)

1. Water Sampling

Samples of 100 liters of river water should be obtained at the water inlet of the plant before any treatment is applied. Unless high turbidity is encountered this volume can be easily sampled with the recommended filters.

Samples of 1000 liters of finished water should be obtained before it reaches the first customers (i.e. after storage in the reservoirs).

2. Virus-adsorbing filters

The best filters for virological analysis of water containing high organic content are the electronegative fiberglass filters. They are less sensitive to the organic and inorganic content of the water.

The recommended filters (10 inches cartridges) are obtained from FILTERITE Corporation Inc. (Timonium,MD):

- prefilter (3 micron porosity) :
  Diamond Filter Tube (cat.no: G-3A-10S)
- a pleated cartridge filter (0.45 micron porosity)
  Duo-Fine Filter (cat.no.: DFN 0.45-10AN)

Cartridge holders, if possible made of polycarbonate (autoclavable), can be obtained from the same supplier or locally. Tubing, connectors, injectors for chemicals, water meters, portable pH meters, etc... are described in the supporting literature. All efforts should be made for maintaining this material virologicaly sterile through chemical (acid wash or chlorine treatment) or heat disinfection.

3. Conditioning of samples:

Raw water samples: before filtration using the proper proportioning methods, the water should be adjusted to pH 3.5+/- 0.2 and 0.001M aluminum chloride. Filtration can be performed at flow rates of up to 20 liters/minute.

Drinking water: before filtration using the proper proportioning methods the water should be dechlorinated with sodium thiosulfate at a rate of 5mg/L of filtered water, adjusted to pH 3.5 +/- 0.2 and 0.001M aluminum chloride. Filtration can be performed at flow rates of up to 40 liters/minute.

Samples should be delivered to the virus lab to be processed the same day.

4. Elution of viruses from filters

The eluent of choice to remove viruses from filters is Beef Extract prepared as a 3% sterile solution adjusted to pH 10 with 5N sodium hydroxide just before use . The elution of the two filters with one pass of the eluent requires 1.5 to 1.7 liters of solution. The elution process should be performed in 3 to 5 minutes by slowly percolating the eluent using a pressure vessel or peristaltic pump. The eluent should be immediately neutralized to pH 7.0-7.5 using 1N hydrochloric acid.

5. Reconcentration procedure

A number of methods have been described for the concentration of the eluate to a final volume of 25 ml or less. Ammonium sulfate precipitation is the latest method described which appears to successfully recuperate most viruses. Essentially, an equal volume of saturated ammonium sulphate (at pH 7) is added to the eluent. The floc that is formed is collected by centrifugation at 4,000xg and resuspended in 10 ml of tissue culture medium (MEM)

6. Decontamination procedure

To eliminate the microbial contaminants in the concentrate, antibiotic are added at a final concentration of 500 units/ml of penicillin, 500 ug/ml of streptomycin, 50 ug/ml gentamycin. The concentrate is then centrifuged at 4000xg for 30 minutes at 20°C and the supernatant carefully collected.

7. Assay procedure

The detection of the enteric viruses should be performed on the most sensitive cell line available. At the moment, the BGM cell line appears to be the cell line of choice. The concentrate obtained in step 6, should be inoculated in a number of flasks containing this cell line. Half of the sample should be inoculated on the first time and the remaining kept frozen for repeats. No more than 1.0 ml/25 cm^2 should be inoculated per flask.
A least one blind passage should be performed on the same cell line with appropriate means of detection of the presence of viruses (immunofluorescence, immunoperoxidase, electron microscopy).

THE EDMONTON DRINKING WATER STUDY:
REPORT AND RECOMMENDATIONS REGARDING
VARIOUS HEALTH EFFECTS QUESTIONS

Prepared for Steve E. Hrudey and Associates Ltd.

by TOXCON CONSULTING LTD.

May 16, 1986

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TERMS OF REFERENCE

The following were the terms of reference for this part of the City of Edmonton Drinking Water Study:

1) Review the recent literature on the following topics and provide overview summary opinions on their application and relevance to potential health effects of Edmonton drinking water:

   a. relationship of water hardness to heart disease.
   b. relationship of sodium to heart disease.
   c. liklihood of risk to the developing fetus from trace contaminants in drinking water.
   d. potential adverse effects of fluoridation at the levels currently used.
   e. relationship of drinking water to Lou Gehrig's disease occurrence in Edmonton.

2) Interview up to 10 respondents to the request for comments to the Water Study who have complained of specific health effects which they attribute to drinking Edmonton water.

3) Based on the foregoing interviews, recommend what follow up action may be warranted to address the health problems of the persons interviewed.

4) Review existing practice within the established health care system in response to individuals who may raise concerns about direct personal health effects of Edmonton drinking water. Advise on any communicative mechanisms which may assist individuals with obtaining help within the existing system.

1. RELATIONSHIP OF WATER HARDNESS TO HEART DISEASE

Despite the tremendous concern in North America with preventing the onset and ultimate effects of cancer, cardiovascular related diseases are still the leading causes of mortality. Cardiovascular disease (CVD) is a general term used to encompass numerous disease groups that involve the cardiovascular system including ischemic (or coronary) heart disease, cerebrovascular disease (or stroke), arteriosclerosis, hypertension, congestive heart failure, myocardial infarction, acute rheumatic fever, chronic rheumatic heart disease et cetera (Smith and Tunstall-Pedoe, 1984).

The relationship between drinking water and health has long been a concern of physicians and public health professionals (Folsom and Prineas, 1982). Since the first studies of John Snow in 1855, demonstrating an association between a "water factor" and the incidence of cholera, many studies have clearly demonstrated cause-effect relationships between waterborne infectious agents and epidemic diseases. These initial findings have had a dramatic effect on our approach to resolving certain environmental health problems. In this regard our heightened awareness of environmental pollution and potential chronic health consequences has led to numerous studies attempting to demonstrate a causal relationship between some inorganic substance in the environment and the onset of a particular health problem.

With regard to drinking water Kobayashi (1957) was the first to suggest that CVD, in particular deaths from cerebral hemorrhage, correlated closely with the characteristics of drinking water in different localities in Japan. This finding set the stage for Schroeder (1960) to examine the association of all deaths including cardiovascular deaths with various water parameters for a number of cities in the U.S. Schroeder found a negative correlation between water hardness and cardiovascular death, that is to say, CVD decreased with increasing water hardness.

Schroeder's report of regional variation in cardiovascular mortality as a function of water hardness led to numerous epidemiological studies designed to explore this relationship in different parts of the world and substantiate it if possible. The most important studies have been conducted in the U.S. and Britain, although studies have also been conducted in Canada, Australia, Finland, France, Ireland, Italy, Japan, the Netherlands, Norway, Sweden, the Soviet Union, Czechoslovakia, the Federal Republic of Germany and other European countries. Reviews of the literature may be found in Heyden (1976), Neri and Johansen (1978), Comstock (1980), Smith and Tunstall-Pedoe (1984), Sonneborn et al (1983). According to Hewitt and Neri (1980) the epidemiological literature alone comprises over one hundred separately published reports in the past 20 years. This does not include numerous biomedical studies conducted with animal models in which various constituents of drinking water have been tested for their ability to induce cardiovascular abnormalities.

In many of these studies the association between water hardness and CVD has been from the perspective of two determinants of hardness i.e. calcium and magnesium levels or from the possibility that "soft water" may dissolve toxic trace metals (e.g. Cd, Pb) from the city distribution systems. These two lines of thought have dominated the direction of both animal and epidemiological research on this issue.

According to Comstock (1980) most of the studies can be discussed in terms of geographic units using five categories of comparisons:

1. all states or major cities within a nation
2. all cities or counties within a state or province
3. all areas within a city or county
4. selected communities within or not within the same unit
5. individual subjects within a defined area.

The first four categories are called "ecological studies" because all variables - water hardness, cardiovascular death rates and other characteristics - are obtained for political units and not individual subjects. Category 5 represents a true epidemiological study because the basic units are persons for whom there are individual estimates of both exposures and outcomes.

In the first category 23 studies (from 1960 to 1976) using various population units within eight different countries support the negative association of cardiovascular mortality with water hardness. The disagreements between studies largely involve levels of statistical significance, methodology and which particular cardiovascular cause of death correlates best with water hardness. It was from these studies that the dictum "soft water, hard arteries" received its initial and strongest support.

In the second category a total of 17 studies conducted between 1964 and 1977 also supported a negative association between water hardness and cardiovascular mortality. However, for these studies the negative association was not as strong as that for category one studies.

In the third category only a few studies have been reported in the literature and these do not support higher death rates with soft water.

The fourth category includes studies of groups of cities that have different levels of water hardness. Of the 11 only 2 favored the soft water, hard artery hypothesis. Six were inconclusive because the differences in water hardness were slight or because the association with water hardness was not significant. Three did not support the hypothesis at all.

With regard to the fifth category very few studies have been conducted. One such study conducted in Canada by Hewitt and Neri (1980) examined the hardness-CVD relationship by examining individual exposures and outcomes in 2 hard water communities (Kitchener, Ontario and Regina, Saskatchewan) where domestic water softeners were used by some of the population. In both communities it was found that there was less cardiovascular mortality in households with water softeners. This clearly contradicts the soft water, hard artery hypothesis!

In the United States 3 studies conducted in Washington County, Maryland took into account the effects of several important demographic, socio-economic and personal characteristics. Two of the studies gave opposite results - an apparent protective effect of soft water for males and a harmful effect for females - while the third study (a 12 year prospective study of 30,000 persons) showed similar discrepant results by sex for deaths from arteriosclerotic heart disease, namely that soft water was associated with lower rates for males and higher rates for females. Comstock concluded that because of these conflicting findings the reality of the association between water hardness and cardiovascular mortality is open to debate.

In order to determine whether an association is one of cause-and-effect or of coincidence epidemiologists use several criteria including:

1. Consistency - Can the finding be replicated by other studies done in different populations, at different places and at different times?

2. Specificity - a given exposure tends to produce a particular type of disease whereas coincidental associations are more nonspecific in their effects. Although specificity favors causality, lack of specificity does not rule it out.

3. Strength of the Association - as measured by the size of the relative risk where large relative risks (eg. 10) come close to guaranteeing causality, but low relative risks do not rule it out. It merely means it is much harder to prove. Furthermore, with a weak association it is difficult to demonstrate a dose-response relationship.

4. Biologic plausibility - i.e. the identification of some component in drinking water that could be implicated in the pathogenesis of CVD or the demonstration that a known risk factor for CVD is also associated with hardness. Again concurrence with known biologic facts favors causality but nonconcurrence does not because of the present gaps in biologic knowledge.

Comstock summarized his personal assessment of the probability of causal association of hardness with cardiovascular mortality as follows:

"The criterion of consistency gives fair support to its causal nature; those of specificity and strength, little if any. I consider that the so-called natural experiments are unconvincing and that only magnesium seems a biologically plausible factor in light of present knowledge. My verdict at the moment is the Scottish one: not proven". The basis of Comstock's conclusion centres on a low relative risk to the individual from drinking softened water and also the questionnable cost-benefit value of undertaking difficult studies to prove a causal relationship between water hardness and CVD.

Although Comstock agrees that further "searches for a water factor are warranted" he strongly advocates the following:

1. the use of exposure measurements from the taps of subjects' homes and not information from the treatment plant.
2. the collection of seasonal samples to establish what the true annual exposure is.
3. the effects on water components of boiling and adding tea, coffee or alcohol.
4. the effects of water composition on the mineral contents of other foods cooked in water.
5. the effects of food components on the absorption of minerals into the body.
6. the establishment of an accurate daily intake of tap water and other fluids.

Comstock argues that studies that ignore these factors run the risk of obscuring any real relationship between a water characteristic and cardiovascular disease. He points out that the advantages of ecological studies of water characteristics and disease are their quickness, their relative low cost, their use of readily available information, their production of useful hypotheses and their tendency to be favored by many scientists in the epidemiologic community. The disadvantages of these studies include the fact that they are rarely definitive and should be considered pilot studies only.

In their review of the literature Hewitt and Neri (1980) report that the yield from much of the epidemiological work has been meagre in terms of agreed upon conclusions. Although they attribute the slow rate of progress in this area on the complexity of the phenomena studied, they site two major deficiencies: (1) a continuing lack of rigorous methods for investigating geographical (and temporal) variations in disease frequency and (2) the rather uncritical use by some investigators of seemingly simple statistical tests, in particular the correlation coefficient.

With regard to the second point Hewitt and Neri indicate that much of the water hardness-CVD literature is questionnable because of the misapplication and overinterpretation of the correlation coefficient. Numerous authors have assumed this measure to be capable of describing a relationship between a geographically varying quantity like water hardness and another geographically varying quantity like local mortality rate. They have also assumed that the statistical significance of such a relationship can be tested by standard methods applied to the correlation coefficient. In the stated context, however, the correlation coefficient has neither of these properties. In fact, "considered as a statistic to be subjected to significance testing, the correlation coefficient - in the context of geographical studies - is virtually useless" (Comstock, 1980).

One of the major shortcomings of the correlation coefficient is that its magnitude is critically affected by the amount of random error in the dependent and independent variables. Thus, correlations based on smaller geographical units (eg. cities) are likely to appear weaker than those based on larger units (eg. provinces or Canada). This is the case with the water hardness-CVD literature where the negative correlation between hardness and CVD is supported in the large national studies but weaker when studied in smaller geographical units. Hewitt and Neri conclude that the only way around this problem is to desist from conducting ecological studies and turn to the study of individual exposures and outcomes. They also recommend the use of the regression coefficient, adjusted for the effects of available pertinent characteristics.

Thus, the correlation coefficient tells only how closely the observations fit a given line and whether the slope is up or down. It fails completely to indicate how strong the association is. In this regard more recent epidemiologic studies have employed the regression coefficient. The verdict from these studies is that drinking water may be a contributory factor in the development of CVD. However, it has been stressed (Calabrese and Moore, 1980; Sharrett, 1981; Shaper, 1984) that CVD is a complex entity affected by age, diet, sex, psycho-social stress, exercise, family background, climate, genetics and other factors. Therefore, the recognition of a multifactorial etiology for CVD is critically important when interpreting the results of studies which do not recognize within their designs the reality of numerous confounding variables. Caution must be exercised before generalizations can be derived from the results of these studies.

Question: Where does this leave us then with regard to Edmonton's drinking water?

By definition water hardness is not a precisely defined chemical characteristic. Rather it is an empirical formulation primarily referring to the multivalent cation content of water. Conversely, softness refers to a relatively low content of multivalent cations. The major cations making up hardness are Ca^{++} (range approximately 1-150 mg/L) and Mg^{++} (range approximately 1-30 mg/L). Generally lesser constituents in approximate descending order of concentration and occurrence are Sr^{++}, Fe^{++}, Al^{+++} and Mn^{++} (McCauley and Bull, 1980).

Edmonton's water is described as "moderately hard" (average total hardness of raw water = 160 mg/L and treated water over past 3 years after deliberate softening by the City = 126 mg/L) which generally fits in with values described in the literature. For example, Masironi et al (1980), in reviewing European evidence, described soft water as < 100 mg/L $CaCO_3$, intermediate water as 100-250 mg/L $CaCO_3$ and hard water as > 250 mg/L $CaCO_3$. By this definition and in comparison with the literature Edmonton could be expected to have a relatively low cardiovascular mortality rate (Masironi et al, 1980). Examination of age-standardized death rates for males in 1983, ischemic heart disease (IHD) in Edmonton ranks fifth in the province behind Jasper National Park (#1), Vegreville (#2), Leduc-Strathcona (#3) and Medicine Hat (#4). It should be noted that Vegreville and Leduc-Strathcona receive drinking water from the City of Edmonton. For cerebrovascular disease in males Edmonton again ranks fifth behind Leduc-Strathcona (#1), Medicine Hat (#2), South Peace (#3) and Alberta East Central (#4). Neither IHD nor cerebrovascular disease was significantly greater than the overall rate for Alberta (Alberta Social Services and Community Health, 1985).

For females the situation is dramatically different. Here Edmonton females rank eighteenth in the province with respect to IHD and fifteenth with respect to cerebrovascular disease. Again neither rate was statistically greater than the overall Alberta rate.

If one compares Alberta to the rest of Canada it is noteworthy that age-standardized death rates for males and females for IHD and cerebrovascular disease have declined markedly in Alberta since 1972 (Newman, 1984). The exact percentages for males are 19% (IHD) and 30% (cerebrovascular disease) and for females 15% (IHD) and 22% (cerebrovascular disease). Furthermore, these death rates parallel the continuing decline of cardiovascular deaths in Canada and yet are still below the national average for each year from 1972-1982, the period examined by Newman.

If one assumes that the composition of Edmonton's drinking water has not substantially changed since 1972 then there is little reason to expect water hardness will result in a significant health risk to the individual with respect to CVD. This point is further strengthened by the finding that many contaminants in drinking water generally provide only a small proportion of the total intake in comparison to the dietary contribution. For example an average of 3% of man's uptake of Cd, Mg, Mo, Fe, Ni and Zn comes from drinking water while 2% I, 2% Mn, and < 1% P, K, Al, Na come from drinking water (Lappenbusch, 1986). Of course, the problem in dealing with averages is that they tend to conceal the extreme values, particularly the peaks and this in itself becomes important when assessing the risk of developing particular diseases such as CVD.

2. RELATIONSHIP OF SODIUM TO HEART DISEASE

2.1 Introduction

It has long been recognized that elevated levels of sodium (Na) exist within many public drinking water supplies in North America. For example, in a survey conducted in 1975 in the U.S. of 630 interstate carrier water supply systems, 42 percent had sodium concentrations greater than 20 mg/L and 3 percent had levels greater than 200 mg/L. However, despite this fact an international standard has not been promulgated because of the lack of definitive human population studies demonstrating adverse health effects of sodium in drinking water. This does not mean, however, that there is no evidence for adverse health effects from the ingestion of high levels of sodium. On the contrary, considerable research has been conducted on animals and humans since the early 1900's. Much of this has focused on the association between sodium intake and hypertension, the dominant health outcome of high sodium intake. It is from this perspective that the American Heart Association (1957) proposed a limit of 20 mg/L to protect those patients with heart or kidney ailments and who require a low sodium diet. The World Health Organization has an esthetic standard of 200 mg/L but no health standard, the U.S. EPA has a monitoring and reporting requirement but again no health standard. It does have a goal of 20 mg/L though for public drinking water (Calabrese et al, 1980). Finally, Health and Welfare Canada have no standard (Lappenbusch, 1986).

2.2 Health Effects of Elevated Sodium Exposure

2.2.1 Animal Research

It is not possible in the time alotted for this report to review critically all the animal studies of the health effects of sodium. Other authors have already reviewed this literature (Calabrese et al, 1980; Folsom and Prineas, 1982) and report the following summary findings for studies with rat, chicken, rabbit, dog and monkey models:

1. Hypertension (i.e. elevated blood pressure) is produced by the chronic ingestion of elevated levels of salt (NaCl).
2. The greater the ingestion of salt, the more severe the hypertension.
3. The younger the animal at the time it is fed a high salt diet, the more sensitive it is to developing rapid hypertension.
4. Genetic factors influence the individual's response to NaCl.

2.2.2 Human Research

Sodium is an essential daily dietary requirement. With suggested daily intakes for the infant, child and normal adult being 430, 1500 and 2200 mg respectively (Lappenbusch, 1986). Typical intakes for North Americans range from 3 to 30 grams with most people in the 5 to 15 gram range (Freis, 1976). Several earlier human studies revealed that salt intake of this order of magnitude resulted in elevated blood pressure levels. In 1922 Allen and Sherill reported that a reduction of salt in the diet could reduce hypertension. Since then numerous epidemiological studies have been conducted confirming the relationship of sodium exposure as a causative factor in the development of hypertension (Calabrese et al, 1980). This conclusion is supported by studies in "uncivilized" populations in Guinea, the highlands of Malaysia, the Easter Islands, rural Uganda and the Kalahari Desert of Africa. These societies have low salt intakes and do not develop hypertension unless they become "civilized".

2.2.3 Evidence Relating Sodium Ingestion Via Drinking Water to Human Health Problems

Since water ingestion contributes only approximately 0.5 to 9.0 percent of the sodium consumed, with the exception of those on restricted salt diets (Schroeder, 1974), most human studies of the relationship between the frequency of hypertension and sodium intake have considered only the contribution of sodium from food and not drinking water.

In one of the first studies on drinking water Furstenberg et al (1941) reported that high levels of sodium in the drinking water resulted in the failure of a low-salt diet to treat Miniere's disease. Elliot and Alexander (1961) reported several cases of recurrent heart failure at home which ceased after substitution of a low sodium drinking water source for a high sodium source. Many other instances such as this exist in the literature. Thus, in individuals on low-sodium diets drinking water can provide a significant proportion of their total sodium intake. For example, Russell (1970) reported that patients on a restricted sodium diet of 500 mg Na/day who consumed 2.5 liters of water/day containing 125 mg Na/L would receive 63 percent of their daily sodium allowance from drinking water. It can, therefore, be seen that high levels of sodium in drinking water may be an important contributing factor in the development of a variety of heart disorders in high risk patients.

Schroeder (1967) suggested that the association between drinking water composition and cardiovascular disease could be an indirect one, mediated through high blood pressure, which itself is a known risk factor for coronary heart disease and stroke. Numerous epidemiologic studies have sought to demonstrate a relationship between drinking water and blood pressure and thereby confirm Schroeder's hypothesis. These have been reviewed by Folsom and Prineas (1982) who analyzed the evidence for several inorganic substances in water including sodium, cadmium, lead, nitrates, and hardness (indicated by calcium magnesium content). They concluded that the evidence was weak, inconsistent, contradictory and insufficient to conclude whether drinking water composition and blood pressure are related. Of the five inorganic variables considered sodium showed the strongest evidence. However, even for sodium only 5 or 6 studies have been performed on a worldwide basis and these suffer from the following deficiencies:

1) not having an a priori hypothesis as a basis of investigation
2) selecting unrepresentative or biased samples
3) failing to use standardized, valid methods of blood pressure measurement and/or water analysis
4) failing to make "blinded" observations where possible
5) incompletely accounting for possible confounding variables related to both consumption of water and blood pressure
6) inadequately characterizing the time and amount of exposure of individuals to the drinking water
7) failure to measure the levels of all major elements simultaneously in samples from the tap not the treatment plant
8) failure to use multivariate statistical analyses to determine the independent effect of each element
9) inappropriate study design

Many of these deficiencies seem to have been resolved in more recent work by Tuthill and Calabrese (1979, 1985). In a carefully designed study these investigators reported the effects of sodium in community drinking water on blood pressure distribution patterns in elementary (third grade) and high school (tenth grade) students in two Massachusett's communities. Their results revealed that the blood pressures of both groups of students from a community with 107 mg Na/L were significantly upshifted as compared to similarly aged students from a closely matched, geographically contiguous community with only 8 mg Na/L. These differences, although very small, were upheld when potentially confounding factors, including dietary sodium intake and other water factors were controlled for in the analysis. It is noteworthy that the total dietary intake of Na in the high Na community was 13% greater than in the low Na one and that about 25% of the excess Na intake in the high Na community came from water.

A subsequent study was designed to test for a dose-response relationship between drinking water Na and blood pressure in two Massachusett's communities with 6 mg Na/L and 42 mg Na/L in drinking water. Results of this study revealed no statistically significant differences in blood pressure between tenth graders from the two communities. The lack of support for a dose-response effect of drinking water sodium and blood pressure was inconsistent with their previous study. The authors suggested that sensitivity of blood pressure to sodium may not be seen until levels of approximately 100 mg Na/L are approached. In this study there were no statistically significant differences in mean Na intakes in males or females between the two communities.

Question: What then are the potential cardiovascular risks from sodium in Edmonton's drinking water?

It is generally accepted that the etiology of CVD, as exacerbated by hypertension, is multifactorial depending on such factors as genetic predisposition, diet, age, socio-economic factors, psycho-social stress changes et cetera. Although numerous variables can influence the blood pressure of an individual the association between drinking water and hypertension (vis a vis blood pressure) is still debatable. Recent epidemiological studies in Massachusetts, USA by Tuthill and Calabrese (1985) suggest that high levels of sodium in drinking water (107 mg Na/L) upshifted blood pressures in elementary and high school students in comparison to a control community at 7 mg Na/L. Under chronic exposure conditions these authors suggest that these students may become hypertensive at an earlier age than expected.

If we exclude high-risk patients at this point, City of Edmonton drinking water data for the past 3 years indicates an average Na level of 4.2 mg Na/L at the treatment plant. If the level of Na coming out of the tap is equivalent to that at the treatment plant, which limited data from Alberta Environment suggests, then the risk to the normal individual would probably be low even if a real association does exist between sodium in drinking water and blood pressure. Given an average water intake of 1.3 L/day for Canadians (Gillies and Paulin, 1983) this would amount to 5.5 mg sodium intake via drinking water which represents 0.1 percent of Lappenbusch's (1986) reported average value of 5656 mg total Na intake/day for North American adults. It should be noted that this calculation does not include the possible increase in sodium content of tap water in homes using Zeolite or NaCl exchange type water softeners.

With regard to individuals who are on sodium-restricted diets (eg. heart or renal patients), it is not possible to assess accurately the potential health risks from drinking Edmonton water without first having specific information regarding the maximum allowable daily sodium intake. For these individuals the level of sodium in the water may still present a hazard and should therefore be considered under the advice of their physician. It should be noted in this case that the concentration of sodium at the tap becomes important, particularly if NaCl exchange type water softeners are used in the home. It is also noteworthy that the standard of 20 mg Na/L set by the American Heart Association (1957) was designed to protect these individuals and the level of sodium in Edmonton water at the treatment plant is 1/4 of that standard. Thus, these patients would appear on the surface at least, to be under no immediate risk, but again, patients should consult with their attending physician.

3. RISK TO THE DEVELOPING FETUS OF DRINKING EDMONTON'S WATER

3.1 Introduction

A review of the literature over the past six years reveals very few epidemiological studies examining the effects of drinking water composition on human fetal development. Of the studies performed most have focused their attention on trace contaminants such as Pb, Se, Zn, Ca and nitrates (Leger and Elwood, 1980; Dorsch et al, 1984; Kundu et al, 1985). A much larger literature base exists, however, for the teratogenic effects of various drinking water constituents in animal models. In the latter case the focus has not necessarily been on establishing an association between drinking water and its potential teratogenic effects. Instead, the focus is on the potential teratogenic effects of a single chemical (regardless of route of exposure) that is being developed for use in the environment (eg. pesticides). In this case drinking water is often chosen as the vehicle for dosing the animal. Nevertheless, in order to assess the risk to the human fetus from chemicals in Edmonton drinking water it is necessary to extrapolate from known teratogenic effects in animal models. This, in itself, is a very difficult task since animals often differ considerably in their handling of various chemicals or chemical combinations.

The City of Edmonton routinely analyzes its raw and treated water for 65 chemicals including pesticides (19), metals (19), organics (11) and inorganic compounds (16) - see Appendix 1. A review of this list reveals the following having teratogenic effects in animals and hence teratogenic potential in humans (NRC, 1977; WHO, 1984; Lappenbusch, 1986):

1) Pesticides/herbicides:
   - Diazinon
   - heptachlor/heptachlor epoxide*
   - endrin
   - 2,4-D
   - 2,4,5-TP

2) Metals:
   - arsenic*
   - cadmium*
   - lead*
   - mercury (inorganic/alkyl)
   - nickel*
   - selenium
   - zinc

3) Organics:
   - phenols

4) Inorganics:
   - chlorine dioxide
   - cyanide
   - nitrates

5) Radionuclides:
   - ^{137}Cs
   - ^{131}I
   - ^{226}Ra
   - ^{90}Sr
   - ^{3}H
   - U

* also evidence for carcinogenicity in animals and/or humans.

3.2 Pesticides/Herbicides

Of the first group, the pesticides/herbicides, examination of 1984 City of Edmonton drinking water data revealed that none of the potentially teratogenic compounds exceeded the maximum acceptable concentrations (MAC's). If one assumes that the data for 1985 and 1986 show similar results, then the potential risk to the developing fetus in the average pregnant female would be low since the health-based MAC's used by the City of Edmonton are international standards that already have safety factors built in. If, however, the pregnant female is hypersensitive to low levels of various chemicals in her environment, it is possible that the fetus may also reflect that sensitivity during its development, in which case drinking water may be a more significant factor in fetal development. Although the frequency of this type of situation is predictably very low, it should not be ignored as a potential contributing factor in cases where malformations do arise. However, proving a cause-effect relationship in this situation would be exceedingly difficult and exposure to other sources of these chemicals would have to be taken into account. For example, exposure could arise from household (eg. plants or controlling pests) or lawn and garden use. Furthermore, the environmental persistence of a given chemical would become a critical factor since compounds with a half-life of hours or days would present less of a hazard than those that are bioaccumulated.

3.3 Metals

With respect to the metals these have received perhaps the most attention from the perspective of potential teratogenic effects. Because many metals are required as essential elements in humans numerous studies have been conducted in animal models to determine not only their toxicity but also their role in the metabolism of the whole organism.

From the aforementioned list arsenic, lead, methyl mercury, nickel and selenium can cross the placental barrier. The potential toxicity to the fetus of these metals will depend upon the solubility of the metal in drinking water, the dose the mother is exposed to by drinking water, the percentage of metal absorption from the gastro-intestinal tract, the final concentration in the maternal circulation, the permeability of the placenta to the metal, the stage of fetal development and the dose at the target site. Of the metals listed on page 11, < 5, 3, 57, <0.01, 3, 8 and 3 percent of the average total human dose respectively comes from drinking water. Thus, lead and selenium would contribute the greatest contribution of total metal uptake by normal adults (Lappenbusch, 1986). An analysis of the average levels of these two elements in Edmonton drinking water (at the treatment plant) for the past three years reveals that they were 1/25 and 1/10 less respectively than their corresponding MAC values. The remaining elements were 1/50 (As), 1/5 (Cd), 1/3 (Hg), no value (Ni) and 1/830 (Zn) of their respective MAC values. Nickel does not have an MAC value because of its low toxicity, its low concentrations in drinking water and the small daily intake of nickel in drinking water (compared to food) - NRC, 1977.

It is noteworthy that in areas in North America that use zinc-galvanized pipes for distributing water, cadmium levels in drinking water are higher, particularly if the water has a low pH (Durlach et al., 1985). This occurs because Cd is a normal contaminant of Zn galvanized pipes or seals and tends to leach out under low pH conditions. The level of cadmium in the water is likely also to be a function of how long the water is in contact with the plumbing, and as a consequence there is likely to be a variation in concentration when the water is drawn at different times of the day from the same tap. The average pH of the water coming out of a very limited sample of Edmonton taps was 8.1(1983), 8.3(1984), 8.2(1985) and 8.0(to April, 1986) - (Alberta Environment data). Given this data and more showing that Edmonton's treated water is low with respect to metal corrosivity (a combination effect of high pH and the deposition of calcium precipitates on pipes), the likelihood of Cd leaching into the system is low. In fact, average levels of Cd have been <0.001 mg/L since 1983. This is < 1/5 of the current MAC value. It is noteworthy that from time to time higher than normal Pb readings in the distribution system have occurred (McClure, 1986 - personal communication). When traced back it was found that the sample had come from a line that was "not frequently used and not flushed before the sample was taken".

From a health perspective only between 5-7 percent of the Cd is normally absorbed from the gastrointestinal tract (WHO, 1984). Absorption is affected by a number of factors including age, calcium, iron, zinc and protein deficiency. Thus, in iron-deficient women, as could occur during pregnancy, up to 20 percent of the ingested Cd can be absorbed. Fortunately the placenta acts as an efficient barrier to Cd. Furthermore, magnesium (Mg) is an effective competitive inhibitor of both Cd and Pb in the human placenta, thereby reducing the concentrations reaching the fetus (Durlach et al, 1985). It is important to point out, however, that during pregnancy Mg levels may be deficient and therefore this "protective" effect may not be sufficiently protective. In this case a greater risk would exist from Pb than from Cd because the placenta is quite permeable to Pb and Cerklewski (1983) found Mg deficiency to increase Pb retention in both mother and fetal rats. The risk to the developing fetus from drinking Edmonton water would be, therefore, predictably low.

With respect to selenium its toxicity can be altered extensively by interactions with sulfate, methionine, cystine, mercury, lead, zinc, cadmium, copper, arsenic and vitamin E but little is known about these interactions. Selenium is essential for domestic animals but the margin of safety is relatively narrow. A review of the literature by Lo and Sandi (1980) reveals that in laboratory animal models Se causes developmental deformities. Similar results have been found for sheep, pigs and cattle grazing on seleniferous rangeland during pregnancy. In humans living in seleniferous areas there is also some evidence, albeit weak, that Se may be involved in adverse pregnancy outcomes (Kundu et al, 1985; Lo and Sandi, 1980). Since the average level of Se in Edmonton drinking water is 1/10 its MAC the risk to the fetus is probably very low.

3.4 Organics/Inorganics

Of the organics that are analyzed for in Edmonton's drinking water phenols are perhaps of most interest from the standpoint of the developing fetus. Phenol contamination of drinking water can come from many sources including wastewaters from the petrochemical industry, numerous industries where phenols serve as intermediates, run-off from agricultural land or as natural degradation products of vegetation. Since the term phenols refers to a class of compounds, it is not possible here to predict the potential human teratogenicity of these compounds without knowing first the exact compound, its concentration and the exposure conditions. Suffice it to say that an analysis of the concentrations of phenols in Edmonton drinking water over the past 3 years reveals average levels that did not substantially exceed the MAC. It is important to note that for several chlorinated phenols, toxicity based limits will be much higher than taste or odour-based limits which means that if water is free from taste and odour it is unlikely to present any direct health risks due to chlorinated phenols (WHO, 1984). However, it must be recognized that absence of odour or taste does not guarantee the safety of drinking water in general. Since the teratogenic effects of many phenolic compounds in humans are unknown, it is not possible to assess the risk to the fetus during times when a noticeable odour or taste does exist in Edmonton water (eg. during Spring thaw conditions).

With regard to the three inorganic chemicals previously mentioned very few human population studies have been conducted to examine test for potential teratogenic effects. Since the chlorination of raw surface waters is known to form carcinogenic trihalomethanes, chlorine dioxide (ClO_2) is now being used in many cities as an alternative disinfectant. Suh et al (1983) designed a study to determine the effects of ClO_2 and its metabolites, chlorite (ClO_2^-) and chlorate (ClO_3^-) on rat fetuses *in utero*. Female rats were administered ClO_2 at 0, 1, 10 and 100 mg/L and ClO_2^- or ClO_3^- at 1 and 10 mg/L daily in drinking water for 2 1/2 months prior to and throughout gestation. In the case of ClO_2 at 100 mg/L decreases in the numbers of successful embryos implanting and live fetuses per dam were found. Increased incidences of skeletal anomalies such as incompletely ossified or missing sternebrae or rudimentary ribs were seen in all treatment groups, but there was no significant difference compared with controls. The authors concluded by stating that ClO_2 in drinking water at 10 mg/L is relatively harmless to rats when fed prior to and throughout gestation. However, chlorite and chlorate at the same concentration increased fetal length. From the few animal studies that have been performed on ClO_2 and its metabolites it would appear that they may have teratogenic effects in more than one species of animal. However, a major criticism of these studies is that the sample size was too small to be able to detect teratogenic effects. At the present time no international standard for these compounds appear to exist. The City of Edmonton has established that the chlorine dioxide and chlorite levels at the high lift pump shall not exceed a 0.5 mg/L limit. Also the operating criteria is to maintain a residual of 0.10 to 0.19 mg/L of ClO_2 in the process at least 30 minutes after addition of chlorine dioxide.

Considering cyanide next it is noteworthy that it is almost always present wherever life and industry occur. Both inorganic and organic forms of cyanide exist with many cyanides forming part of life processes, in particular as metabolic intermediates. The commonest forms of cyanide include hydrocyanic acid (in solution), soluble cyanide salts and metallocyanide complexes.

In water hydrocyanic acid dissociates at about pH 8.2 into the cyanide anion (CN^-) and H+. Conversion of cyanide to the much less toxic cyanate at pH > 8.5. In general, the levels in raw water tend to be low (usually < 0.1 mg/L; WHO, 1984). Chlorination of potable water to a free chlorine residual under neutral or alkaline conditions will reduce the concentration of cyanide in finished waters to low levels.

From a health perspective the cyanide ion is rapidly absorbed and its effects, including inhibition of oxygen utilization by cells, rapidly produced. A single dose of 50-60 mg is fatal to humans but exposures of 2.9-4.7 mg/day are non-injurious to humans owing to the highly efficient detoxification system in which cyanide is converted to the relatively non-toxic thiocyanate ion. On the basis of animal experiments an acceptable daily intake for cyanide is 8.4 mg.

A review of the average levels of cyanide in Edmonton drinking water for the past three years revealed the following: < 0.0025 mg/L (January + February, 1986), < 0.011 mg/L (December, 1985), < 0.0024 mg/L (1984). Thus, the levels recorded were 10 to 100 times lower than the MAC. This should be adequately protective for the developing human fetus. (WHO, 1984).

The final inorganic compounds of interest are the nitrates. Under normalconditions source water nitrate content is below 10 mg/L but concentrations of > 100 mg/L have been observed in some parts of the world. Nitrates get into drinking water from fertilizer use, decayed vegetable and animal matter, domestic effluents, sewage sludge disposal to land, industrial discharges, leachates from refuse dumps, and atmospheric washout. In surface water they tend to get depleted by aquatic plants. Increases in nitrates in water tend to vary seasonally with higher concentrations occurring following the application of nitrogen fertilizers or after a heavy rainfall following severe drought conditions.

Because none of the conventional water treatment and disinfection practices modify levels of nitrate to any appreciable extent, the tap water is often very similar to source waters. A majority of people are exposed to < 5 mg nitrate as nitrogen/L. However, this only accounts for < 0.7 percent of the total daily nitrate ingestion (NRC, 1977). Vegetables (86%) and cured meats (9%) account for the greatest daily nitrate intakes.

The major health concern related to high nitrate exposure of mothers in the formation of nitrites which react with blood hemoglobin to produce methemoglobin, a pigment which cannot carry oxygen. Several studies have been conducted on long-term chronic feeding of nitrite to rats. Of particular interest is a study performed by Guener et al (1973) who showed transplacental passage of nitrite in pregnant rats given doses of 2.5-50 mg/kg nitrite orally or intraperitoneally. This dose produced methemoglobinemia in the fetuses and the result was that the rats born had higher mortality rates and poor growth and development.

In humans the evidence for teratogenic effects of nitrates alone is scarce and from nitrates in drinking water even more scarce. A recent study by Dorsch et al (1984) in rural South Australia investigated the relationship between mothers' antenatal drinking water source and malformations in the offspring. It was prompted by earlier descriptive findings of statistically significant, and localized, increase in perinatal mortality in Mount Gambier, due principally to congenital malformations affecting the central nervous system and multiple organ systems. Compared with women who drank only rainwater during their pregnancy, women who consumed principally groundwater containing 5-15 mg/L nitrate had a three-fold increase in risk of bearing congenitally malformed children while those drinking water > 15 mg/L had a four-fold increase in risk. Despite these findings the authors nevertheless concluded "... given that other unidentified teratogenic factors could be correlated with water supply - either within or outside the water source - it would be premature to interpret our case-control findings exclusively in terms of water nitrate exposure. Nevertheless, we believe that the internal cohesion of our findings, and their consistency with our earlier study and experimental evidence, lend weight to the possibility of a real association between groundwater nitrate consumption and malformations".

A review of the nitrate and nitrite levels in Edmonton drinking water over the past three years reveals average concentrations of 0.102 and 0.05 mg/L respectively. These values are 1/100 (nitrates) and 1/20 (nitrites) the MAC standard followed by the City and, therefore, should present little risk to the developing fetus. However, it is important to emphasize that this area could clearly use more research such as establishing the levels of nitrate/nitrite in tap water over the course of a year. This suggestion arises because of the reported seasonal variation in nitrate levels in surface waters, the ineffectiveness of treatment systems in removing nitrates and the seasonal gradient in risk in congenital malformations reported by Dorsch et al (i.e. highest for spring and summer conceptions and lowest for winter and autumn conceptions respectively).

3.5 Risk From Radionuclides in Edmonton Drinking Water

Minute traces of natural radioactivity are normally found in all drinking water (NRC, 1977). The concentration and composition of these radioactive constituents vary from place to place, depending principally on the radiochemical composition of the soil and the rock strata through which the raw water has passed. Natural radionuclides include potassium-40 (^{40}K), tritium (^{3}H), carbon-14 (^{14}C), rubidium-87 (^{87}Rb), radium-226 (^{226}Ra), polonium-210 (^{210}Po), uranium (U) and its daughters, thorium (Th), radon-220 (^{220}Rn) and radon-222 (^{222}Rn).

The interest in the levels of these nuclides in drinking water stems from numerous studies of external radiation in which it is generally accepted that developing mammals (intrauterine and juvenile) are more radiosensitive than adults (Casarett and Doull, 1975; NRC, 1977). Exposure of the fetus at certain critical times during its development can result in increased tumor incidence, death or developmental abnormalities. The level of fetal exposure depends on the maternal body burden of radionuclides and in particular the concentration of nuclides in the maternal circulation, the ease with which the nuclide crosses the placental barrier and the rate of removal of the nuclide from the circulation.

To some extent all drinking water obtained from surface sources will be contaminated with radionuclides from atmospheric testing of nuclear weapons. However, in these days where testing is being curtailed, the levels of these nuclides has dropped dramatically. Because they account for a major part of the potential dose from nuclear fission and activation products and because of their biological significance, considerable attention has been devoted to strontium-90 (^{90}Sr), cesium-137 (^{137}Cs), iodine-131 (^{131}I), tritium and carbon-14 (^{14}C) as water contaminants.

Another potential source of radionuclide contamination of drinking water is the exhaust air and liquid wastes from universities and medical institutions. NRC (1977) reported that "No evidence yet suggests a drinking water hazard from medical institution effluents", a statement based on several surveys in the U.S. The radionuclides of concern here were ^{131}I and technitium-99m (^{99m}Tc), both which are widely used in medical practice and experimentation.

As previously stated in order for radionuclide to be effective in altering particular developmental processes they must be present at a high enough concentration during the critical period of hours or days at which these processes are occurring. If the dose is low and the exposure protracted over a longer period of time, then this essentially reduces the liklihood of the manifestation of embryotoxic or teratogenic effects.

What then is the risk to the developing fetus exposed to Edmonton drinking water? Lappenbusch (1986) indicates that less than 1 percent of the average amount of radiation to which North Americans are exposed comes from drinking water supplies. NRC (1977) estimated an average background radiation exposure of approximately 100 mrem/person/year of which 0.244 mrem/yr came from drinking water. The U.S. National Council on Radiation Protection (NCRP) recommended in 1971 a maximum radiation dose for fertile women (with respect to the fetus) of 500 mrem/year. Examination of the City of Edmonton drinking water data for 1984 and January, 1986 for levels of ^{3}H, ^{226}Ra, ^{90}Sr, ^{131}I, ^{137}Cs and U indicates that all radionuclides were several times below the maximum acceptable concentrations at the treatment plant. Therefore, the risk to the developing fetus can best be summarized as follows:

> "The developing fetus is exposed to radiation from radionuclides in drinking water for nine months. Thus, the total dose accumulated by the fetus will be very small. Furthermore, although the fetus is sensitive to the effects of radiation in some stages of development, these periods are sharply limited and extremely short. For this reason, too, the total dose administered that could possibly have developmental and teratogenic effects would be extremely small. Current concentrations of radionuclides in drinking water lead to doses of about one five-thousandth of the lowest dose at which a developmental effect has been found in animals. Therefore, the developmental and teratogenic effects of radionuclides would not be measurable" (NRC, 1977).

Again, it should be pointed out that the data analyzed were not from tap samples in Edmonton homes. However, it seems inconceivable that a hazardous level of radioactivity could leach into the City's distribution system. It would, nevertheless, be worthwhile examining levels of the aforementioned radionuclides in tap water in view of the recent Chernobyl nuclear accident.

4. POTENTIAL ADVERSE EFFECTS FROM FLUORIDATION OF EDMONTON DRINKING WATER

4.1 Introduction

The issue of benefits versus potential adverse health effects from the deliberate fluoridation of Edmonton drinking water is perhaps the most difficult to deal with, particularly since one estimate of the number of pro and contrafluoridation publications is about 500,000. Although numerous reviews of the literature exist, perhaps the most useful here are those by NRCC (1977), Clemmesen (1983), WHO (1984) and Smith (1985). It is important to emphasize that the author has attempted to critically analyze the highly contraversial issues surrounding fluoridation of drinking water from an unbiased, toxicological perspective. Obviously, the time alotted for this portion of the project was not nearly adequate to critically analyze the original articles upon which these reviews are based.

An analysis of the reviews reveals three major human health-related issues with respect to fluoridation of public drinking water supplies. These are:

1) dental caries incidence reduction vs development of dental fluorosis
2) dental caries incidence reduction vs fluoride-induced cancer
3) dental caries incidence reduction vs other fluoride-induced illnesses.

4.2 Dental Caries Reduction and Dental Fluorosis

With regard to the efficacy of fluoride at 1 mg/L (drinking water) on the reduction of the incidence of dental caries, a voluminous literature exists demonstrating an average incidence reduction of 50 to 65 percent among persons who have consumed fluoridated water regularly since birth. However, it is also noted in the literature that there is a narrow margin of safety between beneficial effects of fluoride and the development of dental fluorosis (mottling), particularly in young children (0-7 years) and those more sensitive to fluoride (Driscoll et al, 1983; WHO, 1984). In these groups fluorosis may occur at 0.7 to 1.3 mg/L (NRC, 1977).

In late 1985 the U.S. Environmental Protection Agency (EPA) redefined the term "dental fluorosis" from an adverse health effect to a cosmetic effect. They used this redefinition as the basis for increasing the Recommended Maximum Contaminant Level (RMCL) for fluoride in drinking water from 1 mg/L to 4 mg/L, saying mottling, although it could occur in some persons, was not considered an adverse health effect by the U.S. Surgeon General. A Secondary Maximal Contaminant Level (SCML) of 2 mg/L is what the EPA concludes is best for drinking water as it protects teeth from decay but causes no harmful effects. This conclusion is in direct contrast to the Driscoll et al (1983) study that demonstrated moderate to severe dental fluorosis in Illinois communities exposed to 2 to 4 mg/L fluoride and the Hellwig and Klimek (1985) study in Germany reporting a similar finding at 1 to 1.5 mg/L.

Thus, from the standpoint of Edmonton drinking water, it would appear that as long as fluoride concentrations do not exceed 1.2 mg/L (and they did not between 1984-1986) the incidence of dental caries should be lower than in communities (eg. Calgary) that use unfluoridated water and the incidence of dental fluorosis should also be lower. With regard to these hypotheses it appears that no such comparative studies have been conducted in Alberta to test their validity (Alberta Dental Association, 1986).

With regard to the question of sensitivity to fluoride, claims that there are some people who are hypersensitive or allergic to fluoride have generally been dismissed (WHO, 1984). One example cited is the lack of reports from millions of tea drinkers who ingest considerable quantities of fluoride from tea leaves (0.14 mg or 5% of total daily fluoride uptake - NRCC, 1977) without adverse reactions. The WHO concludes, however, that "the possibility of sensitivity or some idiosyncratic reaction cannot be completely dismissed, although from the evidence available the incidence of such cases would be expected to be small". Dean (1946) projected the proportion of a population showing mild fluorosis at 1 mg/L would be about 10 percent. Driscoll et al (1983) confirmed Dean's projection by finding < 15 percent of the children in their Illinois study had dental fluorosis at a level of 1 ppm fluoride in water.

Obviously the risk of dental fluorosis exists for a small proportion of Edmonton residents. The critical decision to be made in whether the benefits from caries reduction for the majority of the population outweigh the risks of dental fluorosis for a minority group. In 1966, the City of Edmonton held a plebiscite on fluoridation of city water and the result was positive. Therefore, the population has made a decision regarding the risks and benefits of fluoridation.

4.3 Carcinogenic Implications

Clemmeson (1983) provided the most recent review of the alleged association between artificial fluoridation of water supplies and cancer. He points out in his article that this alleged association was first presented in 1975 by two biochemists - Dr. John A. Yiamouyiannis and Dr. Dean Burk (a former U.S. National Cancer Institute employee for 35 years) who compared crude cancer mortality rates for 10 fluoridated and 10 nonfluoridated cities in the U.S. Their work has been criticized heavily within the scientific community in many ways and a debate between the antifluoridationists (led by Yiamouyiannis and Burk) and the U.S. Government still appears strong after at least 20 years. Clemmeson concludes from a review of evidence from several countries, including Canada, that this association does not exist and even if it did, it still would not necessarily indicate a causal relationship because of the many interrelating factors.

Given that many human epidemiological studies attempting to establish associations between adverse health effects and drinking water often suffer from design deficiencies, it seems that the most rational verdict on this aspect of the fluoride issue must be the same as Comstock's for cardiovascular disease, i.e. not proven.

It is noteworthy, however, that two recent studies by Tsutsui et al (1984 a, b) show morphological and neoplastic malformations induced by sodium fluoride in cultured Syrian hamster embryo cells and human oral keratinocytes. While genotoxic effects of substances on in vitro culture lines are not direct evidence of carcinogenicity, they should raise suspicions of a compound being a potential carcinogen (Smith, 1985). Coupled with this evidence is research showing that sodium fluoride is mutagenic, causes chromosomal breaks and initiates unscheduled DNA synthesis. In normal toxicological work these are warning signals for the carcinogenic potential of a compound. In the case of Edmonton drinking water, hydrofluosilicic acid is used to fluoridate the water. Thus, the results of Tsutsui et al (1984) with sodium fluoride may not be applicable. Consequently, the fluoride species produced by Edmonton's fluoridation method should be characterized at least for its/their mutagenicity in a properly validated battery of short-term in vitro assays. If results from these tests warrant it, further testing to determine carcinogenic potential may be necessary.

4.4 Other Illnesses

The body responds to its total body burden of fluoride and since fluoride is bioaccumulated in the bone, other fluoride-induced illnesses are often manifest. For example, over long periods of time even very small intakes of fluoride (eg. 1.8 mg/day) lead to raised levels of fluoride in bone (Smith, 1985). This can result in osteosclerosis. Furthermore, as an antagonist of iodide, fluoride may interfere with the physiological function of the thyroid gland and lead to pathological developments within this organ (eg. goiter formation).

Another example would be persons suffering from excessive thirst such as in diabetes insipidous or poorly controlled diabetes mellitus. In these cases the individual may drink large quantities of water, resulting in acute and/or chronic fluoride intoxication. Chronic effects on kidneys, especially in individuals with long-standing kidney disease or polydipsia, has also been observed. People who are on long-term hemodialysis will constitute a particular "at risk" group, especially if fluoridated water is used.

Finally individuals that have a sub-optimal nutritional status, especially with regard to calcium, magnesium, vitamin C, manganese or low Ca/P ratio, suffer toxic effects from fluoride exposure more readily.

From the standpoint of Edmonton drinking water it is very difficult to assess the frequency of these illnesses that may be exacerbated or caused by fluorides.

5. RELATIONSHIP OF EDMONTON DRINKING WATER TO LOU GEHRIG'S DISEASE

Lou Gehrig's disease or amyotrophic lateral sclerosis (ALS) is an uncommon motoneuronal degenerative disease characterized by progressive wasting and weakness of the bulbar and spinal musculature. It has an age-adjusted incidence of 1.8/100,000 white Americans or Canadians (Melmed and Krieger, 1982). Its epidemiology is poorly understood as is its etiology, although with respect to the latter viruses, metals, endogenous toxins, immune dysfunction, endocrine abnormalities, impaired DNA repair, altered axonal transport and trauma have all been linked with ALS. Convincing research evidence of a causative role of these factors is yet to be demonstrated (Bradley and Krasin, 1982; Tandan and Bradley, 1985). In view of this situation it is not possible at this time to ascribe the frequency of ALS in Edmonton to the drinking of Edmonton's water. Simply not enough information exists.

6. CONCLUSIONS

On the basis of a review of the literature over the past 6 years and analytical data for Edmonton drinking water from 1984-1986 the following conclusions have been reached:

1) The association between water hardness and cardiovascular disease is still debatable and a cause-effect relationship has still not been proven.
2) Since soft water or low pH may increase levels of Cd and Pb in plumbing systems and these elements are known to cause adverse health

effects, it would appear inadvisable at this point to "soften" Edmonton drinking water for health or economic purposes beyond the degree to which the City is already softening the water.

3) On the basis of Alberta and Canadian statistics Edmonton does not appear to have a significantly higher incidence of cardiovascular disease.

4) No firm association or cause-effect relationship exists between levels of Na in drinking water and the incidence of heart disease although it appears that individuals on sodium-restricted diets may be at a greater risk of CVD. For the rest of the population sodium is unlikely a significant risk factor since its levels in raw water are about as low as they get in natural water (McClure - personal communication).

5) The concentration of Na in Edmonton drinking water should, therefore, be considered a potential risk factor to renal and heart patients and individuals on low-Na diets, particularly in homes that use NaCl exchange type of water softeners.

6) The risk to the human fetus from exposure to Diazinon, heptachlor/heptachlor epoxide, Endrin, 2,4-D or 2,4,5-TP alone can only be extrapolated from animal studies and therefore the validity of a 1:1 extrapolation is open to debate. For the average female the risk from drinking water contaminated with any one of these chemicals would be quite low. However, the risk to a hypersensitive pregnant mother and fetus could be much higher if the hypersensitivity is genetically determined. For fetuses exposed to a combination of all of these chemicals, the risk is unknown because of insufficient information in the literature on possible additive or synergistic effects of mixtures of these chemicals.

7) Of the metals analyzed in Edmonton drinking water Pb and Se would present the greatest risk to the developing fetus since a higher proportion of the total daily intake of each is derived from drinking water. However, since the average levels of each of these metals over the past 3 years has ranged from 1/3-1/25 less than their respective MAC's, the hazard to the fetus should be relatively low. This assumes, however, that the levels of these metals are the same in all tap water as they are at the treatment plant which appears to be the case from a very limited analysis of tap water since 1983.

8) The effect of the degree of softness and pH of Edmonton's drinking water on the levels of metals in tap water has not been sufficiently characterized since water from only a few taps has been routinely analyzed.

9) The teratogenic potential of phenols in Edmonton drinking water cannot be assessed at present because the exact phenols, their concentrations and exposure conditions have not been characterized in tap water. Furthermore, little information exists in the literature about the teratogenic effects of phenols in humans.

10) ClO_2 may have teratogenic effects in mammals. On the basis of the very low concentrations of ClO_2 residue that characteristically exist in Edmonton drinking water, the risk to the fetus would probably be low.

11) Trace amounts of cyanide exist in Edmonton's raw water. Since the levels recorded in treated plant water are 10 to 100 times below the current MAC, the risk to the developing fetus is low but obviously not zero.

12) The greatest risk to the fetus from maternal nitrate uptake is related to their conversion to nitrites and subsequent oxidation of hemoglobin to methemoglobin. In a developing fetus this could create a hazardous situation. To date no firm association has been established between nitrates in drinking water and the induction of congenital malformations. Furthermore, at the present levels of nitrates and nitrites observed in Edmonton drinking water a hazard is unlikely to exist.
13) Exposure of the fetus to radionuclides at certain critical times during its development can result in teratogenic effects. Levels of ^{3}H, ^{226}Ra, ^{90}Sr, ^{131}I, ^{137}Cs and U in Edmonton drinking water for 1984 and January, 1986 indicate that all nuclides were several times lower than their MAC's. Therefore, the risk to the developing fetus would probably be very low.
14) The incidence of dental caries in populations exposed to 1 mg/L fluoride in drinking water from birth is significantly lower than in populations where fluoride levels are lower. Therefore, from the standpoint of caries prevention it appears that 1 mg/L fluoride in water may be beneficial, although studies to support this statement in Alberta are lacking.
15) Dental fluorosis appears to occur in 10-15 percent of individuals (so called "sensitives") exposed to 1 mg/L fluoride in drinking water from birth. The severity of fluorosis in these individuals is usually mild.
16) The association between increasing incidence of cancer in humans and fluoride exposure is still not proven. Neither is a cause-effect relationship. However, in two mammalian cell lines in vitro tests show sodium fluoride induces neoplastic malformations. The applicability of these findings to Edmonton's drinking water situation is unknown at this point since the City uses hydrofluosilicic acid and not sodium fluoride to fluoridate its water.
17) Higher levels of fluoride in the body appear to induce other illnesses such as osteosclerosis and thyroid malfunction or may exacerbate more long-term diseases such as diabetes insipidous, diabetes mellitus, polydipsia, other kidney ailments. The effect(s) of fluoride in Edmonton drinking water on these conditions has not been determined.
18) The etiology of Lou Gehrig's disease is suggested to be multifactorial but no convincing evidence exists yet as to the association of any drinking water constituent and the disease. Therefore, it is not possible at the present time to relate the incidence of Lou Gehrig's disease in Edmonton to drinking the water.

7. RESPONDANT INTERVIEWS

In late 1985 an advertisement was placed in the Edmonton Journal requesting input from the public regarding concerns over Edmonton drinking water. Of the approximately 50 respondents, 10 were randomly selected for personal interviews to clarify in more detail their specific health concerns. Of the 10 respondents TOXCON CONSULTING LTD. was only able to obtain interviews with 8 of these. Two had relocated to unknown addresses.

7.1 Summary of Responses

The following is a summary of the responses to specific questions:

1) Number females = 7 (mean age = 60 yrs; age range: 43-72)
Number males = 1 (age = 45)
2) Ethnic origin: all Caucasian
3) Mean weight: females = 63 kg (range 57-73 kg)
male = 95 kg
4) Current Occupation: females = 6 at home, 1 nun
male = investment consultant
5) Mean number years in present residence:
females = 17 (range: 3-25)
male = 6 months
6) Present drinking water supply:
municipal (no filters/distilling) = 4
municipal (+ filter or distilling) = 2
bottled = 2
soft water = 0
7) Number of respondents complaining of physical ailments after drinking water = 8
8) Number using salt during food preparation or at table = 6
9) Number on physician-restricted diets = 1 (for salt)
10) Number using antibiotics in past year = 2
11) Number using water pills (diuretics) = 1 (1 pill/day)
12) Number using commercial laxatives = 1
13) Number using commercial antacids = 1 (same person as in #12)
14) Number often under tension = 1
15) Number with diabetes = 1
16) Number with history of heart disease in family = 3
17) Number with history of cancer in family = 5 (mostly breast cancer)
18) Number of smokers = 2 (not > 1 pack/day)
19) Number on long-term drug therapy = 3 (one for control of allergies, arthritis and muscle spasms; 1 for diabetes; 1 for hypothryoidism)
20) Number with significant medical problems in past year = 3 (arthritis / pinched nerve; headaches / bladder problems; parasites / middle ear infections)
21) Respondent's perception of present health: fair = 1
good = 4
excellent = 3
22) How often respondents worry about their health: never = 2
rarely = 5
sometimes = 1
23) Number having some adverse health symptoms in Spring = 3
24) Number with allergies = 6 (types include adhesive tape, house dust, chocolate, pollens, cigarette smoke, fumes from household cleaners, animals, perfumes, toiletries and dermatitis from water)
25) Number reporting reproductive problems = 0
26) General activity level of respondents: light housework = 5
office = 1
athletics = 2
27) Number using nutritional supplements = 6

28) **Beverage usage**
  a) alcohol: infrequent (< monthly) = 5
    light (weekly or monthly) = 3
    moderate (1/day) = 0
    heavy (> 1/day) = 0
  b) coffee/tea: infrequent (< 1 cup/day) = 4
    light (1-3 cup/day) = 3
    moderate (3-5 cup/day) = 0
    heavy (> 5 cup/day) = 1
  c) soft drinks: daily = 0
    weekly = 1
    monthly = 3
    never = 4
  d) fruit juice: daily = 4
    weekly = 1
    monthly = 0
    rarely = 3

7.2 Conclusions

From the above information the following general conclusions can be made:

1) Generally, the respondents perceived that they were presently in good to excellent health, and hence, tend to worry about their health rarely.
2) All respondents complained of experiencing some health problems as a result of drinking Edmonton water either this Spring or in the past.
3) Of the respondents 6 had a history of allergies. This may mean that these individuals are more sensitive to adverse health effects from contaminants in Edmonton drinking water. This should be explored in more depth.
4) No reproductive problems were reported, primarily because all females were beyond the child-bearing stage of their lives.
5) Alcohol, coffee/tea and soft drink usage was infrequent to light in all cases. Therefore, the doses of chemical contaminants in each of these sources will be quite low (eg. fluorides in beer and tea).
6) Smoking does not appear to be a contributory factor in the health complaints since only 25% of the respondents smoke.
7) Drugs do not appear to be a confounding variable here because only 2 individuals are on long-term drug therapy and symptoms do not persist when bottled water is substituted for Edmonton drinking water.
8) Age may be a factor in the health effects experienced by the respondent group since the average age of the women was 60 years, an age at which the frequency of gastrointestinal and other symptoms may increase. This could be explored in greater depth using a larger population sample with all age groups represented.
9) The average length of time by females in their present residence was 17 years yet no one indicated that their health problems had persisted that long. It is possible that it takes a long latent period for health symptoms to develop but

the male respondent experienced symptoms after only 6 months in his present residence. Therefore, the role of time is unknown at this point.

10) Generally, the respondents were interested in preventative health measures through the use of mineral/vitamin supplements, exercise, avoidance of certain foods/activities known to be risk factors for various diseases.

It is important to emphasize the limitations of the above analysis. It is based upon a biased and very small sample and cannot be extrapolated to the general population. At best it helps characterize some of the main health concerns and the general health status of the individuals expressing those concerns. It also could help in the formulation of hypotheses to test in more rigorously designed epidemiological studies should these be considered necessary.

7.3 Recommended Follow-Up Actions

The following follow-up actions are recommended to address the health problems of the persons interviewed:

1) Each of the 8 persons interviewed should have their tap water tested for at least the main parameters listed in Appendix 1. This should be performed at regular intervals over the next year to account for seasonal variations and at different times of the day to account for daily variation (eg. in the morning before running water and again after flushing of the pipes has occurred).
2) Because of the high frequency of allergies in the respondent group, each person should be tested by an allergist for their reaction to water, particularly next Spring during the run-off period.
3) Each of the 8 persons interviewed and members of their immediate family residing in the same household should be monitored over the next year for the onset of any health symptoms that may be attributable to something they have eaten or drank.
4) The type of water distribution system inside and outside of each person's home should be characterized and used in conjunction with water analyses to determine whether the distribution systems are increasing the levels of any contaminants.

7.4 Response to Individual's Concerns

Generally speaking if people experience health problems they attribute to their drinking water, they either approach their family physician or take it upon themselves to purify their water at their taps or drink pre-purified bottled water. The feeling by the majority of respondents was that it is hard to deal with medical doctors because of a preconception that "it is all in their head". Other comments were that a lack of facilities existed to test household water, that it was hard to penetrate the bureaucrarcy or that a standard "no problem" response is always received from the City.

As previously seen in this report such concerns are often very difficult to deal with on a strictly scientific basis since the literature is often confusing, contraversial or insufficient with respect to specific health effects from drinking water. Unfortunately, in many cases engineers have assumed the responsibility of interpreting epidemiological or toxicological findings to the average citizen. This situation is intensified when maximum allowable concentrations are put into place since any excursion beyond a given MAC is seen by the public as a hazardous situation.

It is important to emphasize also that in many cases of environmental pollution health effects can be expressed subtly at the subclinical level (eg. as a change in enzyme activity) or as a subjective symptom such as tiredness that a physician would not recognize as environmentally-related. Furthermore, these effects may be masked or exacerbated by differences in lifestyle, age, sex, genetic predisposition to adverse responses et cetera.

In dealing with public health concerns over drinking water it is essential that both physicians and representatives of the City or the Alberta Government be objective and collect as much information as possible, preferably through a standardized questionnaire. There are numerous instances in the literature where good listening skills and follow-up have uncovered more serious environmental health problems or resolved politically contentious issues in a rational manner. Obviously an integral component in dealing with such concerns is the use of a multidisciplinary approach where engineers, administrators, toxicologists and physicians get together with the common purpose of resolving the problem.

Another mechanism that could prove beneficial in dealing with water issues is the development of a more comprehensive public education program by the City. Specifically, the public should be made aware of what chemicals are being monitored, why they are being monitored, results of the monitoring program including publication of instances when MAC's have been exceeded and why. Such a program might also have a 24 hour telephone number (perhaps a hot-line) where health complaints can be lodged and this should be advertised widely, particularly during Spring run-off. These suggestions should be explored in more detail.

From the standpoint of the individual he/she should be willing to contact local authorities with their concerns and participate in programs or efforts designed to ensure the safety of drinking water. This may mean paying more in taxes or using less biocides or fertilizer but the alternative (i.e. contaminated water) is unacceptable.

8. SUMMARY AND RECOMMENDATIONS

A review of water quality data for the City for the past 3 years and recent scientific literature concerning the topics outlined in the Terms of Reference of this report indicate that, in general, from a toxicological perspective drinking Edmonton's treated water imposes little risk to its citizens. This conclusion is based on the assumption that the water taken from the taps of consumers is not substantially different with respect to its composition from water analyzed at its two treatment plants. Limited data from Alberta Environment suggests that this assumption is probably valid although further research on a larger sample of Edmonton consumers would be useful in clarifying this point.

Of the ten recommendations listed below the author feels that numbers 1, 8 and 11 should be given priority in terms of immediacy, particularly #8 and #11. While the author feels the other recommendations are also important, less sense of urgency is attached to them. The following recommendations are therefore made:

1) The greatest single problem in assessing the association between some drinking water constituent and a potential adverse health effect is the use of contaminant levels measured at the treatment plant as representative of what individuals are being exposed to at their taps. Since the quality of water in the distribution system may be altered from the treatment plant to the tap, it is recommended that water samples from a limited but representative cross-section of consumers be collected and analyzed. Furthermore, this should occur seasonally for the next year to account for any seasonal variations and daily to account for any daily variations in contaminant levels (eg. in the morning before line flushing and again after line flushing). This work should be contracted out by the City to a university or private contract laboratory.
2) In order to determine whether specific areas of the City encounter more quality changes than others, it is also recommended that the results in #1 be correlated against the type of material the distribution and household systems are composed of.
3) It is recommended that research be supported to determine the effects on Edmonton water composition of boiling and adding tea, coffee or alcohol since respondents drinking boiled water reported a decline of their health problems.
4) It is recommended that research be supported to determine the effects of Edmonton water composition and nature of the cookware used on the mineral contents of "essential" foods cooked in water, specifically Mg and Ca which are implicated in CVD.
5) In order to be able to establish the contribution of drinking water to the total daily intake of various constituents, it is recommended that research be supported to establish the daily intake of water and other fluids in Edmonton.
6) It is recommended that a limited survey be conducted with heart and renal specialists to determine whether they experience any irregularities with high risk patients drinking Edmonton water, especially with respect to sodium and fluoride effects in heart and renal patients.
7) In light of the recent Chernobyl, USSR nuclear energy plant catastrophe, it is recommended that the City's raw water be monitored for all medically-relevant radionuclides and that the results be made public as soon as available. Again, this work should be contracted out to a university or private contract laboratory.
8) It is recommended that the City support research designed to elucidate more clearly the effects of fluorides in its

drinking water, beginning with the characterization of fluoride species in Edmonton water and followed by the performance of a properly validated battery of toxicity tests.

9) If the results from studies performed in #8 warrant it, then the deliberate fluoridation of Edmonton drinking water should be re-evaluated.
10) It is recommended that the City of Edmonton not soften their water to a degree greater than is currently practiced in view of the reported, yet unproven, association between soft water and a higher incidence of cardiovascular disease.
11) The recommended follow-up actions for the 8 individuals complaining of health problems resulting from drinking Edmonton's water be adopted.
12) The City explore ways to make the public more aware of what chemicals it monitors routinely, why these chemicals are monitored and the results of this monitoring.

9. BIBLIOGRAPHY

Alberta Dental Association. Personal communication. 1986.

Alberta Social Services and Community Health. Birth and Death Statistics by Local Health Authority. 1983. Alberta SSCH, 1985.

Allen, F. and J. Sherill. The treatment of arterial hypertension. J. Metab. Res. 2:429-545. 1922.

Bradley, W. and F. Krasin. A new hypothesis of the etiology of amyotrophic lateral sclerosis. The DNA hypothesis. Arch. Neurol. 39:677-680. 1982.

Calabrese, E. and G. Moore. Conference on cardiovascular disease and drinking water factors - principal findings and future needs. J. Environ. Pathol. Toxicol. 4:323-326. 1980.

Calabrese, E. and R. Tuthill. The Massachusetts blood pressure study, Part 1. Elevated levels of sodium in drinking water and blood pressure levels in children. Tox. Ind. Health. 1(1):1-10. 1985.

Calabrese, E., R. Tuthill, T. Sieger and J. Kar. The role of elevated levels of sodium in diet and drinking water on the development of hypertension in animal models and humans. J. Environ. Pathol. Toxicol. 4:143-150. 1980.

Casarett, L. and J. Doull. Toxicology: The Basic Science of Poisons. L.J. Casarett and J. Doull (eds). Macmillan Publishing Co. Inc. New York. 1975.

Clemmesen, J. The alleged association between artificial fluoridation of water supplies and cancer: a review. Bull. World Health Org. 61(5):871-883. 1983.

Comstock, G. The epidemiologic perspective: water hardness and cardiovascular disease. J. Environ. Pathol. Toxicol. 4:9-25. 1980.

Dean, H. Epidemiological studies in the United States. In: Dental Caries and Fluorine. Moulton, F.R. (ed). American Association for the Advancement of Science, Lancaster Science Press. pp. 5-31. 1946.

Dorsch, M. et al. Congenital malformations and maternal drinking water supply in rural South Australia: a case-control study. Am. J. Epidem. 119(4):473-486. 1984.

Driscoll, W. et al. Prevalence of dental caries and dental fluorosis in areas with optimal and above-optimal water fluoride concentrations. JADA. 107:42-47. 1983.

Durlach, J., M. Bara and A. Guiet-Bara. Magnesium level in drinking water and cardiovascular risk factor: a hypothesis. Magnesium 4:5-15. 1985.

Elliott, G.B. and E. Alexander. Sodium from drinking water as an unsuspected cause of decompensation. Circulation 23:562-566. 1961.

Folsom, A. and R. Prineas. Drinking water composition and blood pressure: a review of the epidemiology. Am. J. Epidem. 115(6):818-832. 1982.

Freis, E. Salt, volume and the prevention of hypertension. Circulation 53(4):589-595. 1976.

Furstenberg, A., G. Richard and F. Lathrop. Meniere's disease: addenda to medical therapy. Arch. Otolaryngol. 34:1083. 1941.

Gillies, M. and H. Paulin. Variability of mineral intakes from drinking water: a possible exploration for the controversy over the relationship of water quality to cardiovascular disease. Int. J. Epidem. 12(1):45-50. 1983.

Gruener, N., H. Shuval, K. Behroozi, S. Cohen and H. Shecter. Methemoglobinemia induced by transplacental passage of nitrites in rats. Bull. Environ. Contam. Toxicol. 9:44-48. 1973.

Hellwig, E. and J. Klinek. Caries prevalence and dental fluorosis in German children in areas with different concentrations of fluoride in drinking water supplies. Caries Res. 19:278-283. 1985.

Hewitt, D. and L. Neri. Development of the 'water story': some recent Canadian studies. J. Environ. Pathol. Toxicol. 4:51-63. 1980.

Heyden, S. The hard facts behind the hard-water theory and ischemic heart disease. J. Chron. Dis. 29:149. 1976.

Karppanen, H. Ischaemic heart disease: an epidemiological perspective with special reference to electrolytes. Drugs 28 (Suppl. 1):17-27. 1984.

Kobayashi, J. On geographical relationships between the chemical nature of river water and death rate from apoplexy. Berichte des Ohara Institute fur Landwirtschaftliche Biologie 11:12-21. 1957.

Kundu, N., P. Parke, L. Petersen, I. Palmer and O. Olson. Distribution of serum selenium, copper and zinc in normal human pregnancy. Arch. Env. Health 40(5):268-273. 1985.

Lappenbusch, W. Contaminated Drinking Water and Your Health. Lappenbusch Environmental Health, Inc. Alexandria, Virginia, U.S.A. 1986.

Leger, A. and P. Elwood. Neural tube malformations and trace elements in water. J. Epidem. Comm. Health 34:186-187. 1980.

Lo, M-T and E. Sandi. Selenium: occurrence in foods and its toxicological signficnce - a review. J. Environ. Pathol. Toxicol. 4:193-218. 1980.

Masironi, R., Z. Pisa and D. Clayton. Myocardial infarction and water hardness in European towns. J. Environ. Pathol. Toxicol. 4:77-87. 1980.

McCauley, P. and R. Bull. Experimental approaches to evaluating the role of environmental factors in the development of cardiovascular disease. J. Environ. Pathol. Toxicol. 4:27-50. 1980.

Melmed, C. and C. Krieger. A cluster of amyotrophic lateral sclerosis. Arch. Neurol. 39:595-596. 1982.

National Academy of Sciences, National Research Council, Safe Drinking Water Committee. Drinking water and Health. Volume 1. Washington, D.C. National Academy of Sciences. 1977.

National Research Council of Canada. Environmental Fluoride, 1977. Ottawa, Ontario, Canada. 1977.

Neri, L. and H. Johansen. Water hardness and cardiovascular mortality. Ann. N.Y. Acad. Sci. 304:203. 1978.

Newman, S. A perspective on the health of Alberta. Alberta Social Services and Community Health. December, 1984.

Russell, E. Sodium imbance in drinking water. JAWWA 62(2):102-105. 1970.

Schacter, M. Adverse clinical effects of fluoride. National Fluoridation News - Fall. 1984.

Schroeder, H. Relationship between mortality from cardiovascular disease and treated water supplied. Variations in states and 163 largest municipalities of the United States. J. Am. Med. Assoc. 172:1902-1908. 1960.

Schroeder, H. Cadmium, chromium and cardiovascular disease. Circulation 35:570-582. 1967.

Schroeder, H. The role of trace elements in cardiovascular diseases. Med. Clin. North Am. 58:381-396. 1974.

Shaper, A. Geographic variations in cardiovascular mortality in Great Britain. Br. Med. Bull. 40(4):366-373. 1984.

Sharrett, A. Water hardness and cardiovascular disease. Circulation 63(1):247A-250A. 1981.

Smith, G. Fluoride, teeth and bone. Med. J. Australia 143:283-286. 1985.

Smith, W. and H. Turnstall - Pedoe. European regional variation in cardiovascular mortality. Br. Med. Bull. 40(4):374-379. 1984.

Sonneborn, M., J. Mandelkow, D. Schon, and H. Hoffmeister. Health effects of inorganic drinking water constituents, including hardness, iodide and fluoride. Critical Reviews in Environmental Control 13(1):1-22. 1983.

Suh, D., M. Abdel-Rahman and R. Bull. Effect of chlorine dioxide and its metabolites in drinking water on fetal development in rats. J. Appl. Tox. 3(2):75-79. 1985.

Tandan, R. and W. Bradley. Amyotrophic lateral sclerosis: Part 2. Etiopathogenesis. Ann. Neurol. 18:419-431. 1985.

Tsutsui, T. et al. a. Sodium fluoride-induced morphological and neoplastic transformation, chromosome aberrations, sister chromatid exchanges, and unscheduled DNA synthesis in cultured Syrian hamster embryo cells. Cancer Res. 44:938-941. 1984.

ibid b. Induction of unscheduled DNA synthesis in cultured human oral keratinocytes by sodium fluoride. Mut. Res. 140:43-48. 1984.

Tuthill, R. and E. Calabrese. Elevated sodium levels in public drinking water as a contributor to elevated blood pressure levels in the community. Arch. Environ. Health. 34(4):197-203. 1979.

Tuthill, R. and E. Calabrese. The Massachusetts blood pressure study, Part 2. Modestly elevated levels of sodium in drinking water and blood pressure levels in high school students. Tox. Ind. Health. 1(1):11-17. 1985.

World Health Organization. Guidelines for Drinking-water Quality. Volume 2. World Health Organization. Geneva. 1984.

APPENDIX 1

Parameter

Aldrin + Dieldrin
Antimony
Arsenic
Barium
Benzene
Bicarbonate
Boron
Cadmium
Calcium
Carbaryl
Cesium 137
Chlordane, Total Isomers
Chloride
Chlorine Dioxide Residual
Chlorine (free)
Chromium, Total
Copper
Cyanide
DDT, Total Isomers
Diazinon
Diclofop-methyl
Endrin
Ethylbenzene
Ethylene dibromide
Fluoride
Heptachlor + Heptachlor Epoxide
Hexane
Iodine 131
Iron
Lead
Lindane
Magnesium
Manganese
MCPA
Mercury
Methoxychlor
Methylchloride
Methyl Parathion
Nickel
Nitrate (as N)
Nitrite
Nitrilotriacetic Acid
Nitrite (as N)
O-xylene
Parathion
PCB's
Pentane
Phenols
Potassium
Radium 226
Selenium
Silver
Sodium
Strontium 90
Sulphate
Sulphide
Toluene
Toxaphene
Treflan
Triallate
Trichlorophenol
Trihalomethane
Tritium
Uranium
Zinc
2,4-D
2,4,5-TP

A CRITICAL ASSESSMENT OF DRINKING WATER IN EDMONTON

TASK 2.2 Microbiological Characteristics

Prepared for

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by

Daniel W. Smith & Associates Ltd.
Edmonton, Alberta

June, 1986

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1. Introduction

The problems associated with the delivery of very good quality water from a river source is highly dependent on the quality of the raw water, on the treatment processes and their organization, and on the degree of effort exerted in controlling the processes for optimum system performance. This Task Report addresses one part of the first component, the raw water microbiological quality.

1.1 Background

The North Saskatchewan River at the City of Edmonton receives flow contributions from a drainage basin of about 27,970 square kilometres (1). The River flows from its headwaters in the Columbia Icefield through the sparsely populated east slope foothills of the Rocky Mountains to the City of Edmonton. Rural agricultural activities, impoundments, Boreal forest drainage, power plant and industrial discharges and wastewater from a few communities contribute to the water quality of the North Saskatchewan River at the west boundary of the City.

A total of 227 outfalls discharge to the North Saskatchewan River or its tributaries within the City (2). Of these, 29 are combined trunk sewers which transport dry-weather flow to the Gold Bar Wastewater Treatment Plant. During wet-weather overflow goes to the river. The 198 storm sewers carry stormwater and spring snowmelt runoff to the river. Cross connections and pumpwell overflows during wet-weather flow conditions add sanitary sewage to an additional 43 of the storm sewers (3).

From the west boundary of the City to the Rossdale Water Treatment Plant intake location a total of 85 discharges to the river system have been identified. These are summarized in Table 1.1 (2,3).

Table 1.1 Summary of Outfalls to the North Saskatchewan River Within City That Are Up-Stream of the Rossdale Water Treatment Plant*

Storm outfalls which receive overflow from sanitary and combined system	22
Outfalls from combined sewer system	7
Storm outfall with no sanitary overflow	56
TOTAL	85

*Developed from references 2, 3 and 4

1.2 Terms of Reference

This report responds to Task 2.2 of the master project entitled "A Critical Assessment of Drinking Water in Edmonton." Task 2 involved evaluation of the source of Edmonton's raw water supply, namely the North Saskatchewan River, in relation to existing water quality criteria and other raw water sources for major comparable cities in Canada, the U.S. and Europe. Component Task 2.2 relates to the microbiological characteristics. The specific task descriptions are as follows:

1. Assemble data on the microbiological quality of the North Saskatchewan River upstream of Edmonton's water intakes and of any major upstream sources of microbiological contamination. Sources to be reviewed shall include:

City of Edmonton data
Alberta Environment data
Environment Canada data
University studies

2. Organize and analyze data to indicate seasonal and annual trends.
3. Compare data for Edmonton with two or more comparable situations in North America and in Europe.
4. Report on the likely contributions of identifiable sources to raw water quality making appropriate use of river modelling methods.
5. Report on the implications of existing raw water quality to the assurance of microbiologically safe finished water.
6. Propose options for improvement of intake microbiological water quality at E.L. Smith and at Rossdale.
7. Outline any proposed studies which may serve to clarify and, if necessary, improve the margin of safety with regard to microbiological quality of Edmonton drinking water.

2. Microbiological Water Quality Data

2.1 Government of Canada

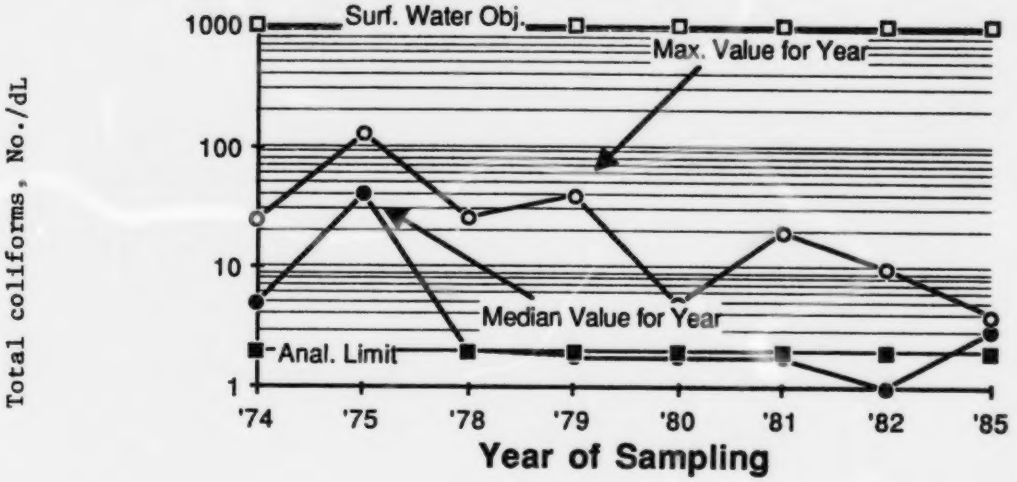
A considerable number of surface water quality data are stored in a national water quality data base, NAQUADAT (5). Table 2.1 provides a brief overview of the bacteriological data available for several NAQUADAT sampling stations on the North Saskatchewan River upstream of Edmonton. Of interest are the low densities of the common indicator bacteria in the waters above Edmonton. Review of selected documents reporting NAQUADAT data (6,7,8,9) reveals a historical trend of near pristine water conditions upstream of Devon. Figure 2.1 presents the results of samples collected at two locations

Table 2.1 Bacteriological Data for NAQUADAT Stations Upstream of Rossdale Water Intake

Station No.	Location	Date	Total* Coliforms	Fecal* Coliforms	Fecal* Streptococci	Remarks
00AL05DA0002	Saskatchewan River Crossing Hwy. 93	1974-75	9(13,<2,23)♦	<1(10,0,*2)	ND†	
00AL05DA0001	Whirlpool Point	1978-82 1985-86	4(54,<1,40) 3(4,0,4)	<2(54,<1,10) <1(4,0,4)	5(53,<1,80) <2(4,0,42)	
00AL05DC1000	Below Bighorn Dam	1985-86	<1(4,0,1)	<1(4,0,1)	2(4,0,3)	
00AL05DC2050	Ancona	1985-86	25(4,6,46)	<2(4,0,5)	6(4,2,13)	
00AL05DC2058	Above Rocky Mountain House	1985-86	56(4,28,296)	23(4,4,58)	41(4,28,71)	
00AL05DC2070	Above Baptiste River	1985-86	44(4,14,196)	13(4,2,36)	84(4,13,146)	
00AL05DD1500	Hwy. 598 Bridge	1983-84	80(3,76,240)	1(3,0,23)	ND	
00AL05DC2090	Above Brazeau River	1985-86	71(4,10,106)	16(4,0,28)	58(4,0,194)	
00AL05DE1000	Drayton Valley Bridge	1983-86	40(7,5,280)	4(7,0,10)	45(4,24,580)	
00AL05DE1500	Genesee Bridge	1985-86	42(4,20,98)	9(4,2,17)	47(4,21,252)	
00AL05DF0008	Devon	1982-84	20(22,<2,300)	6(29,<2,95)	12(9,2,80)	
00AL05DF2067	Devon	1984 1984	48(3,40,80) 32(3,20,68)	4(3,4,48) 4(3<4,12)	208(3,160,212) 192(3,136,216)	left bank right bank
00AL05DF2069	Above Devon Bridge	1982-86	56(25,8,450)	<10(25,<2,120)	20(25,<2,350)	left, right, centre composite
00AL05DF2073	Rabbit Hill	1984	80(3,24,90)	8(3,4,12)	112(3,104,172)	
00AL05DF2075	Above E.L. Smith Pump House	1982	40(11,10,290)	18(11,2,60)	50(11,2,400)	left, right, centre composite
00AL05DF2076	E.L. Smith	1984 1984	16(3,12,80) 60(3,16,152)	<4(3,<4,8) <4(3,<4,12)	116(3,80,196) 124(3,60,180)	left bank right bank
00AL05EB2121	Above Rossdale	1982 1982 1982	350(12,30,5500) 70(12,30,540) 315(12,36,2400)	52(12,10,1000) 16(12,2,46) 66(12,10,170)	65(12,20,370) 37(12,4,270) 65(12,10,210)	left bank centre right bank

*expressed as No. per dL ♦ Median value (no. of samples, min., max.) † not done

(a) Total coliforms from Whirlpool Point



(b) Fecal coliform from Whirlpool Point

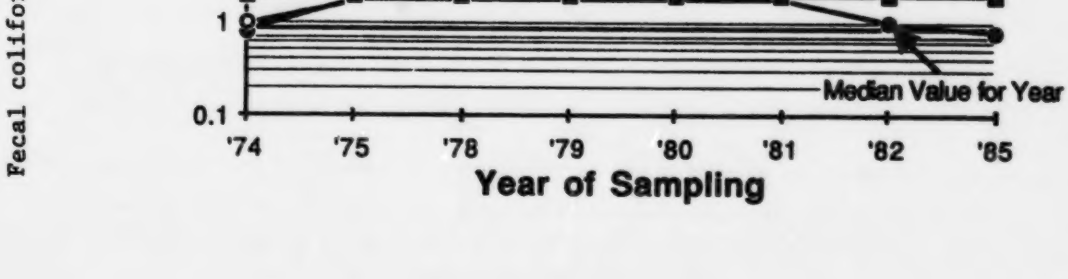
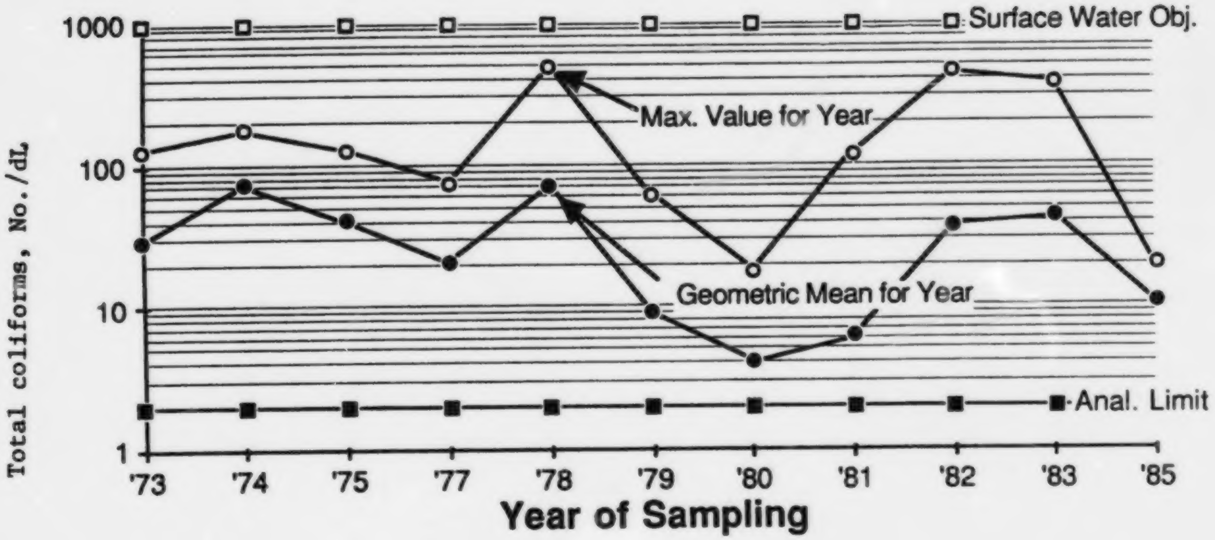
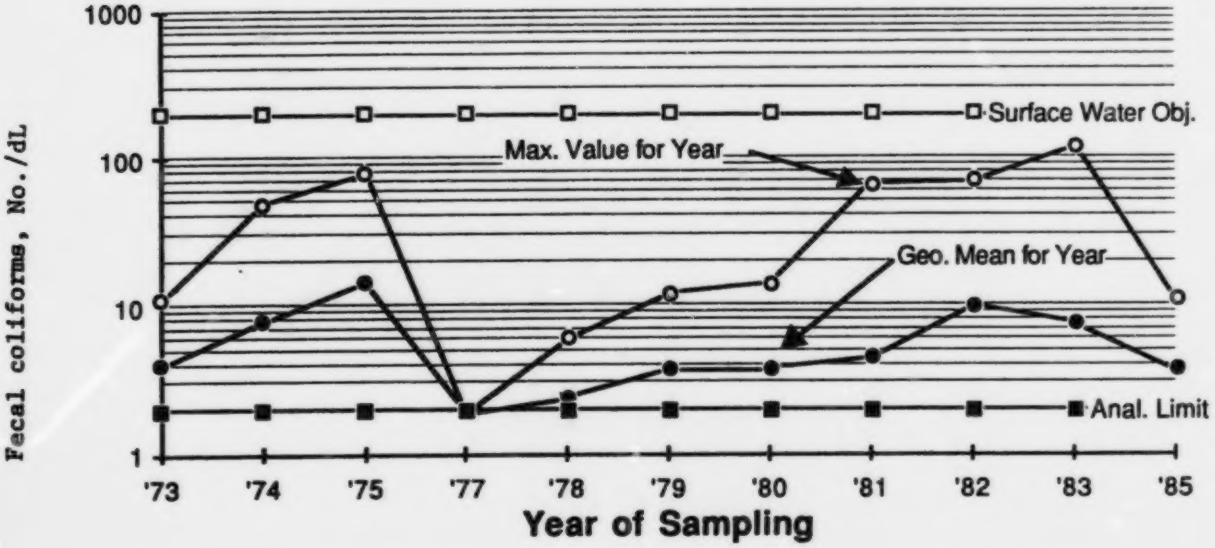


Figure 2.1 Total and fecal coliform values upstream of Edmonton (a) total coliforms from Whirlpool Point, (b) Fecal coliforms from Whirlpool Point, (c) total coliforms from Devon, and (d) fecal coliforms from Devon.

(c) Total coliforms from Devon



(d) Fecal coliforms from Devon



upstream of Edmonton: Whirlpool Point and Devon. The samples analyzed from Whirlpool Point show little change in total and fecal coliforms over the period from 1974 to 1985. In both cases the values reported are near the analytical limits. The data collected from Devon also shows little change in geometric mean values of total and fecal coliforms over the period from 1973 to 1985.

Two reports by Environment Canada (10,11) studied the bacteriology of the North Saskatchewan River in the vicinity of Edmonton.

Table 2.2 (11) summarizes the geometric mean data for the reach from upstream of the Gold Bar Wastewater Treatment Plant to approximately the site of E.L. Smith Water Treatment Plant which was collected in 1975 (identified as Sector I). Table 2.3 (10) summarizes geometric mean data collected in 1974. This document included the research from upstream of the wastewater treatment plant to approximately the 109 Street bridge (also identified as Sector I).

2.2 Government of Alberta

Alberta Fish and Wildlife Division prepared pollution surveys of the North Saskatchewan River and other rivers in the late 1960's (12,13). Upstream of Edmonton, the North Saskatchewan River was reported as having a clear water ecosystem which was stable throughout the year. Studies by the Department of Health in 1969 and 1970 (14,15) and Alberta Environment in 1971 (16,17) revealed sparse data on the bacteriology of the North Saskatchewan River upstream of Rossdale water intake. These are presented in Table 2.4 Stanley Associates (18) sampled the North Saskatchewan River monthly from January to May, 1975. Their data are presented in Table 2.5.

Other studies (19,20,22) confirm the general trend of good water quality upstream of Edmonton, with increasing densities of total and fecal coliforms within the City limits.

Table 2.2 Summary of Microorganism data collected in 1975 for Sector I (11)

Location In River	Total* Coliform	Fecal* Coliform	Fecal* Streptococci
left bank	570	53	20
centre	330	26	11
right bank	530	48	20

* No. per dL, geometric mean

Table 2.3 Summary of Microorganism data collected in 1974 for Sector I (10)

Location in River	Total* Coliform	Fecal* Coliform	Fecal* Streptococci
left bank	307(10,6800) ‡	25(8,1400)	21(<10,630)
right bank	406(20,7600)	70(4,1100)	26(8,140)

*No. per dL, geometric mean ‡ No. per dL (min., max.)

Table 2.4 Early North Saskatchewan River data upstream of Edmonton 1968-72 (14-17)

Station No.	Location	Total* Coliforms	Fecal* ‡ Coliforms
NS2	Drayton Valley Bridge	39	12
NS3	Devon Bridge	40	13
NS4	105 Street Bridge Edmonton	152	12

*No. per dL, geometric mean. ‡ E. coli 1968-70

Table 2.5 North Saskatchewan River Indicator Bacteria Data, 1975 (18)

Location	Total* Coliform	Fecal* Coliform
Devon, right bank	54(11,130)	34(4,93)
Quesnel Bridge, right bank	131(79,240)	43(13,200)
Below Whitemud Creek, right bank	38(0,920)	24(0,540)
centre	17(0,79)	11(0,33)
left bank	123(23,540)	20(0,130)
University of Alberta cooling water outfall	700(170,>2400)	182(33,920)
182 m below outfall	255(0,>2400)	52(0,540)

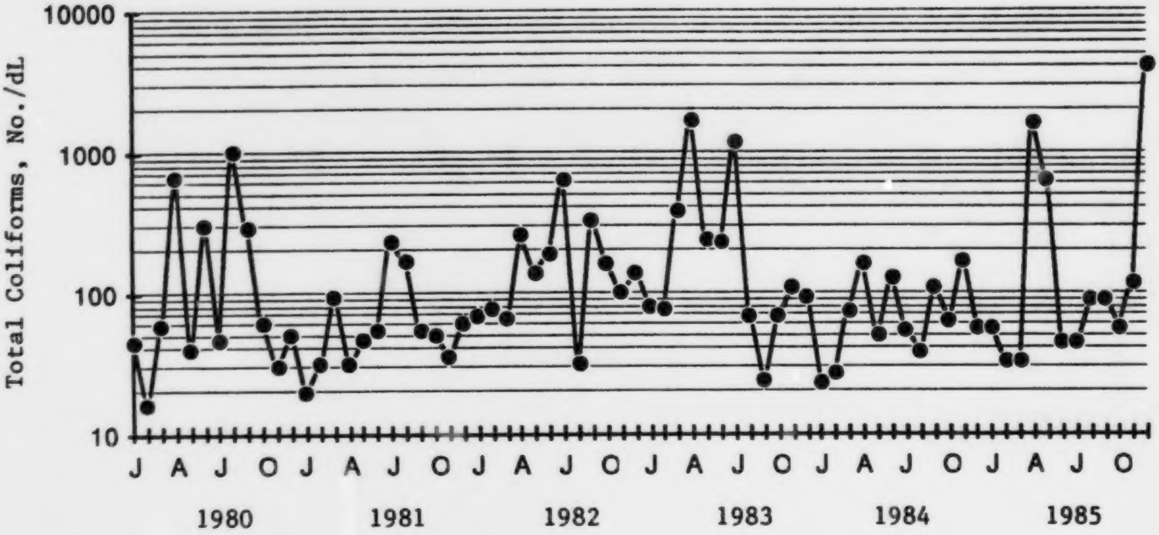
*No. per dL, geometric mean and range

2.3 City of Edmonton

The City of Edmonton has a regular sampling program at the raw water intakes for E.L. Smith and Rossdale Water Treatment Plants. In recent years, total coliform enumeration has generally been done daily and fecal coliforms weekly. Based on reported practice and experience this does not appear to be the best practice for detection of fecal contamination of the raw water source. The data from 1980 to 1985 has been compiled (23-28) and presented graphically for each plant in Figures 2.2 and 2.3. Statistical analysis provides the following confidence interval for the geometric mean total and fecal coliform levels after correction for lag one serial correlation.

E.L. Smith	total coliform (No. per dL)	$62 < \mu < 141$
	fecal coliform (No. per dL)	$5 < \mu < 13$
Rossdale	total coliform (No. per dL)	$501 < \mu < 1047$
	fecal coliform (No. per dL)	$107 < \mu < 224$

The City of Edmonton ceased determining total coliforms as of December 1985. Currently only the results of weekly raw water sampling for fecal coliform analyses are being reported.



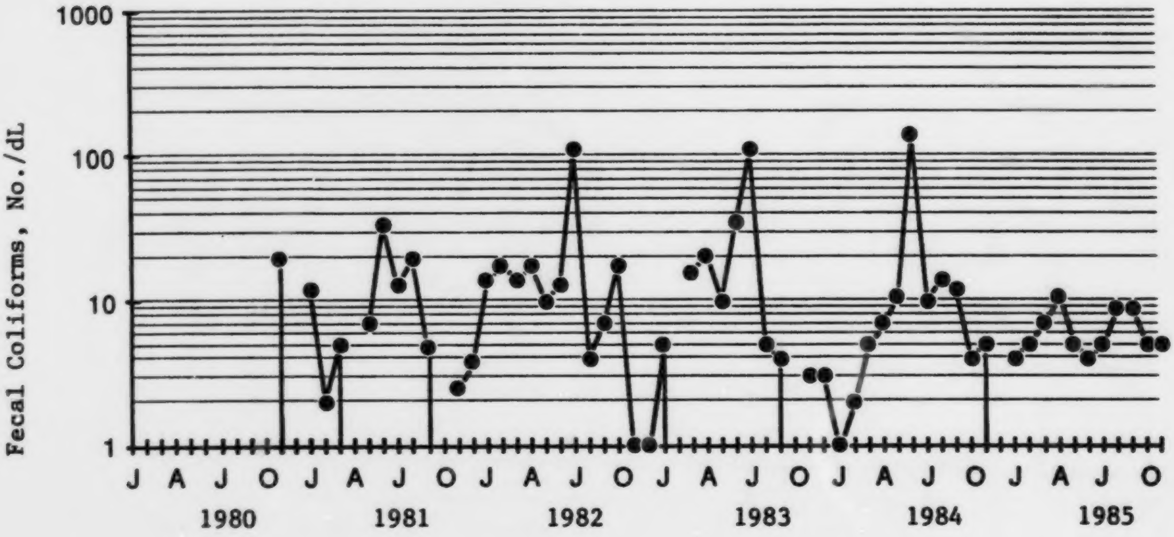
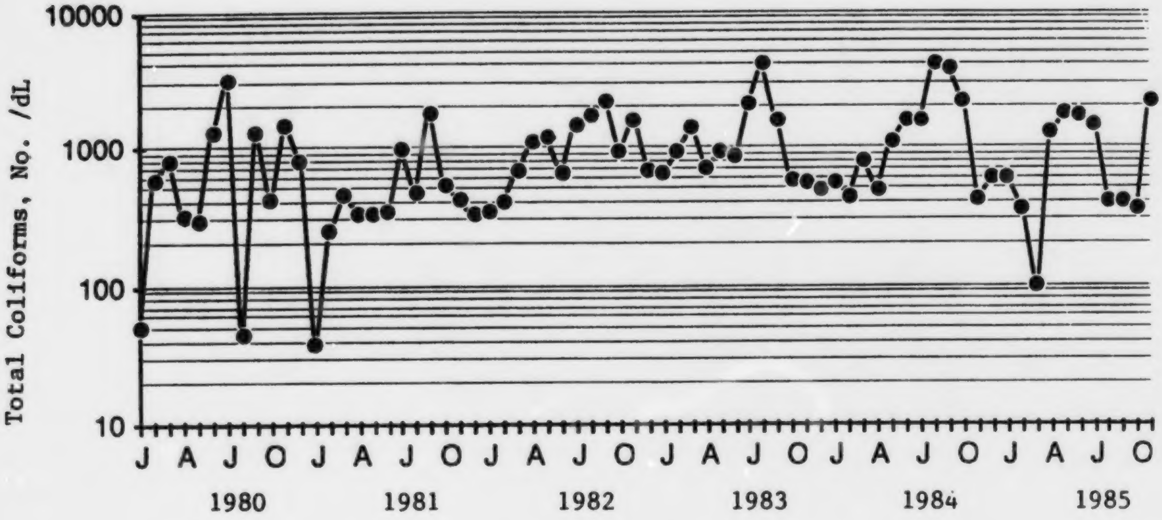


Figure 2.2 E.L. Smith Raw Water Intake, 1980-85



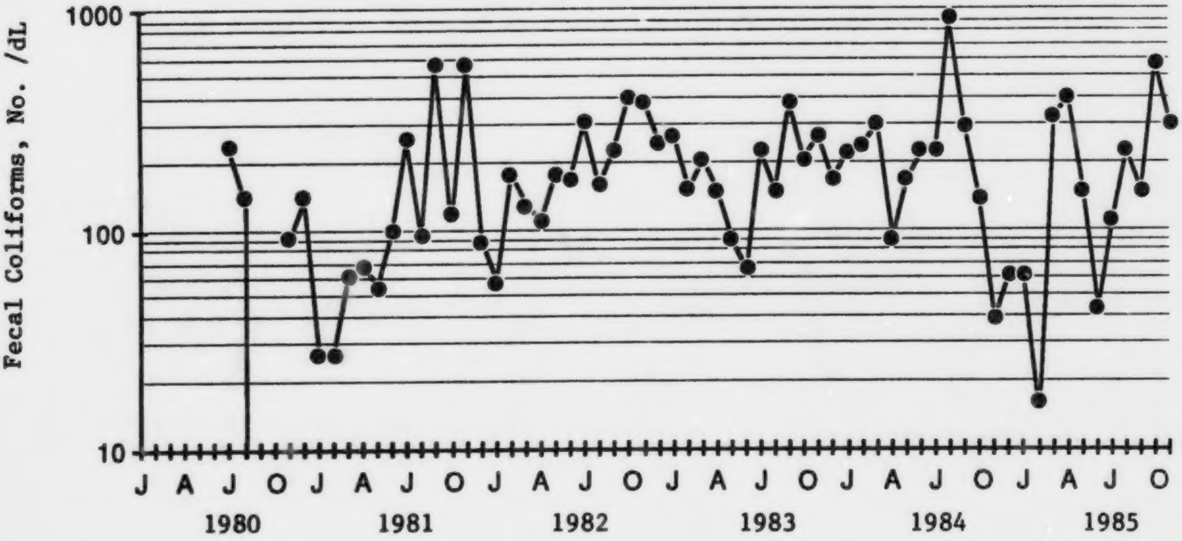


Figure 2.3 **Rossdale Raw Water Intake, 1980-85**

Recent information indicates that 80.4 percent of communities with a population larger than 50,000 perform bacteriological monitoring of their raw water (29). Since the data was not available for those using a surface water supply versus those using groundwater, information on this fact cannot be provided. It is expected that nearly 100 percent of those using surface water sources perform bacteriological monitoring.

During late February and early March 1986, the City of Edmonton intensively sampled significant points in the reach of the North Saskatchewan River between E.L. Smith and Rossdale. The results are presented in Figures 2.4 to 2.7. The sample collection locations were as follows:

- E.L. Smith raw water intake approximately mid-stream.
- Groat Road Sewer outfall (No. 32) discharge.
- University of Alberta water intake structure, approximately 26 m from right bank.
- Rossdale water intake, at left bank (Raw #2).
- Rossdale cross section, 30.5 m from the left bank (Raw #1).

It is clear that significant fecal pollution occurs between E.L. Smith and Rossdale.

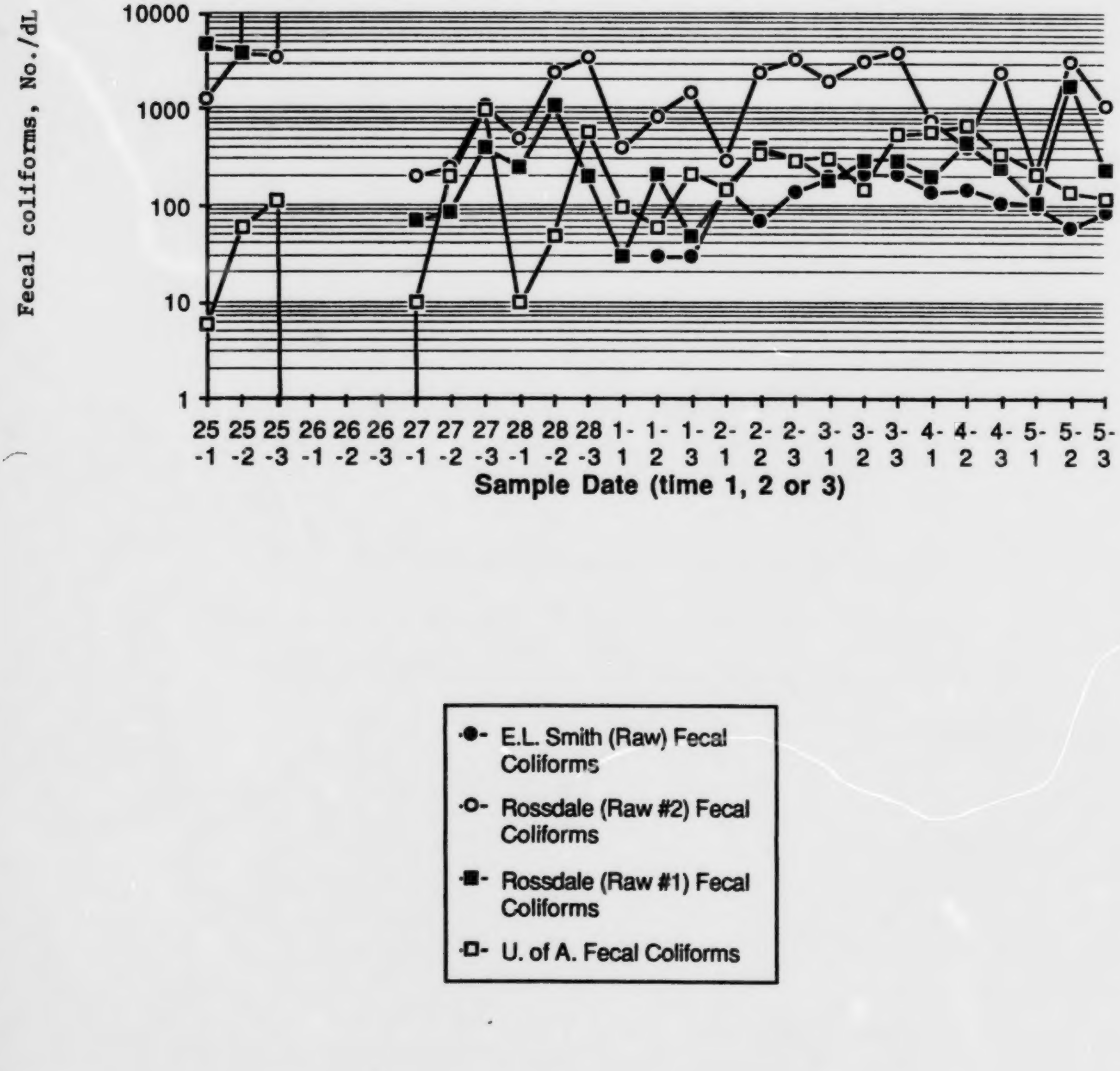


Figure 2.4

Fecal Coliform Counts From Samples Collected At Four Water Intakes, 1986.

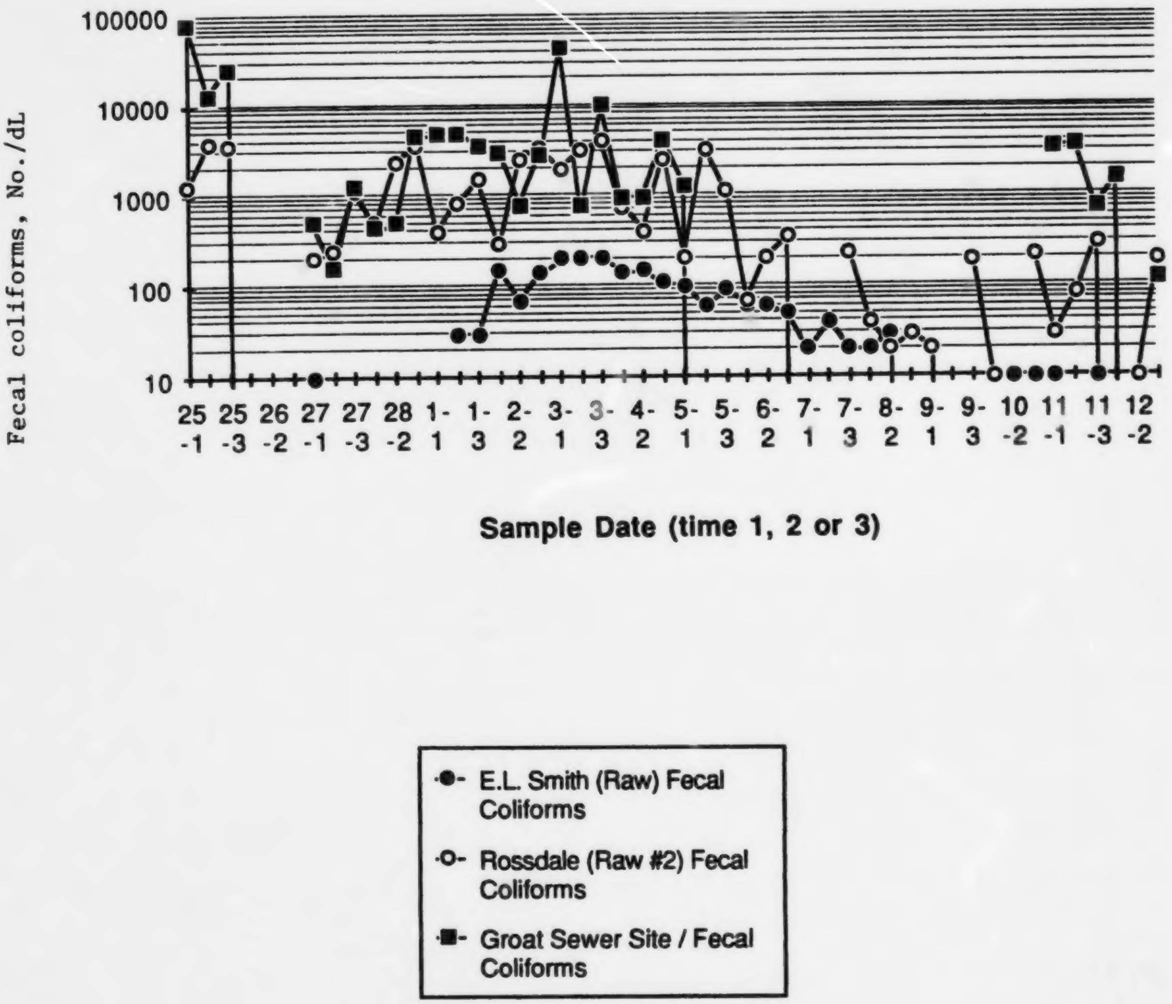


Figure 2.5

Fecal Colilform Counts in Samples From Rossdale and E.L. Smith Intakes

and the Groat Road Outfall, 1986.

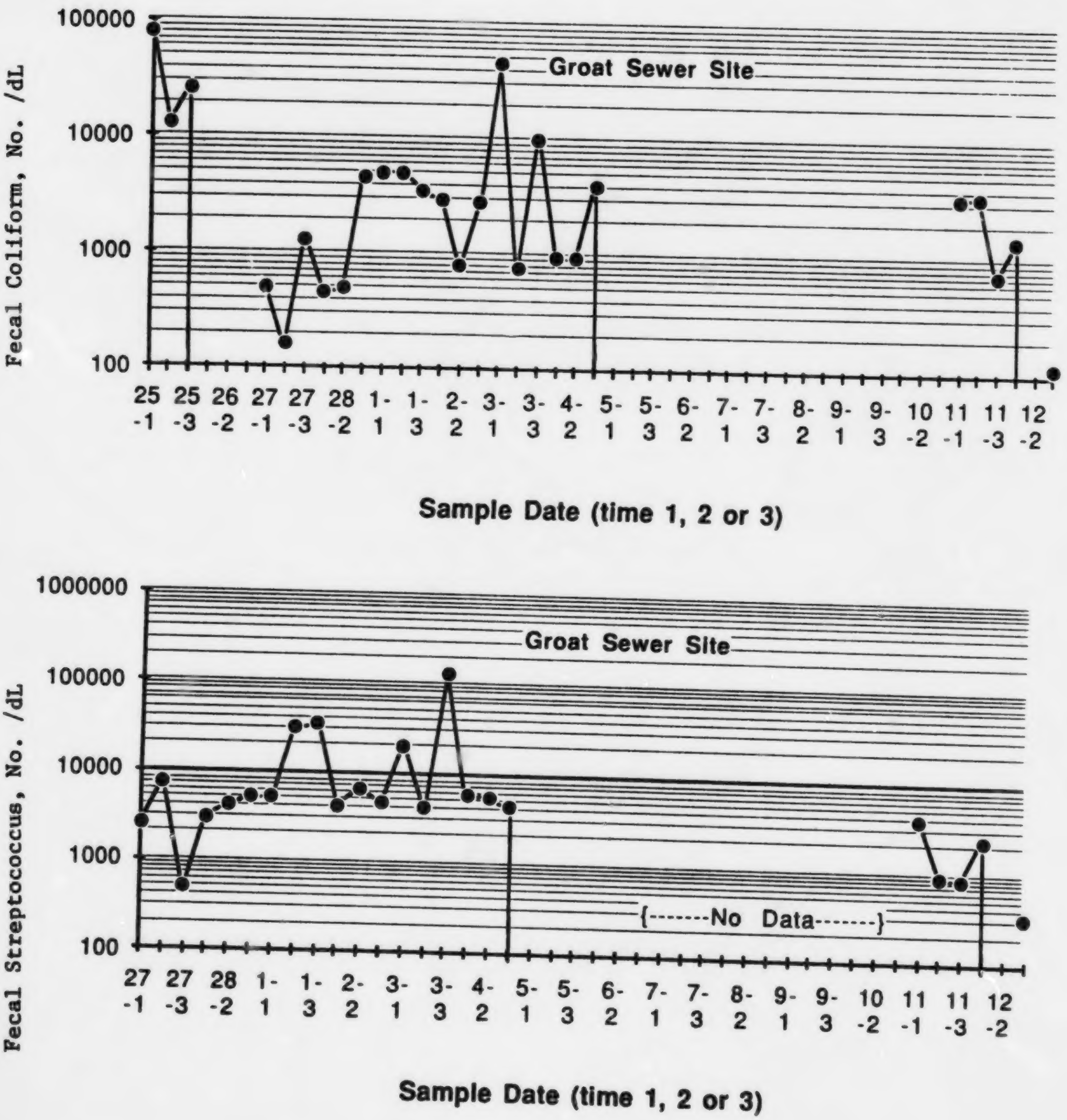


Figure 2.6

Fecal Coliform (a) and Fecal Streptococcus (b) Counts From Samples Collected From The Groat Road Outfall, 1986

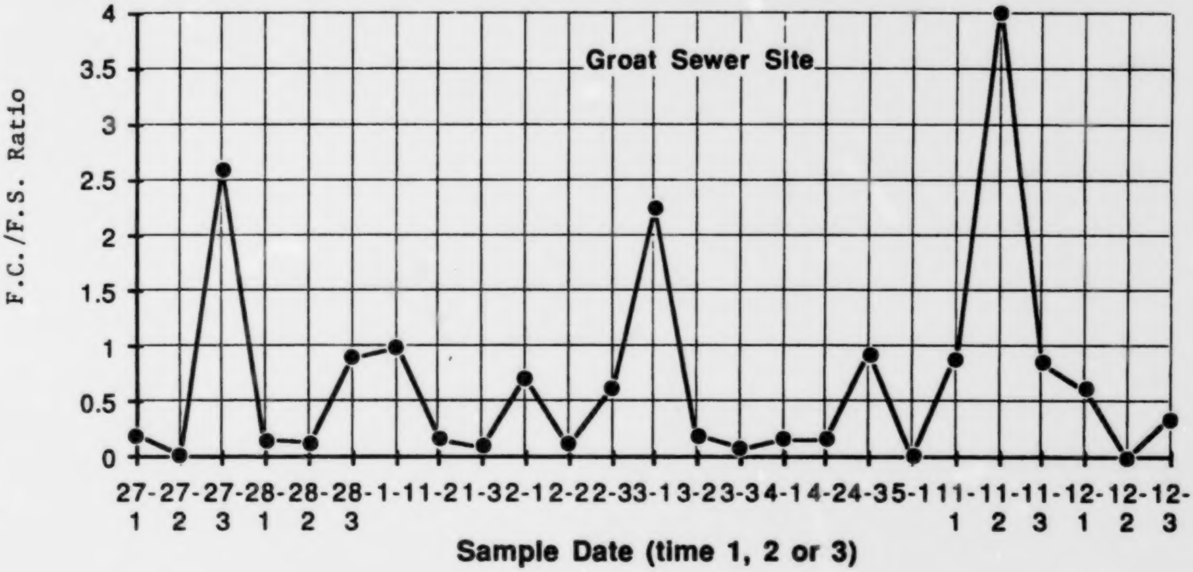


Figure 2.7

Fecal Coliform To Fecal Streptococcus Ratio

For The Groat Road Outfall, 1986

2.4 Studies by Other Organizations

The Edmonton Journal (30) published a series of articles in the fall of 1983 concerning the water quality of the North Saskatchewan River. However they reported elevated levels of fecal coliforms upstream of Rossdale. Unfortunately the data collection and presentation was difficult to interpret properly.

3. Standards and Objectives

3.1. Review of Standards/Guidelines

Health and Welfare Canada (31) has no bacteriological limits on raw water because they believe drinking water technology is sufficiently advanced to treat even heavily polluted water sources. However, they do proceed to define the type of treatment processes required for a given raw water quality as follows:

"1. If more than 10 percent of the raw water samples in any 30-day period have a fecal coliform density greater than 100 per 100 mL or a total coliform density greater than 1 000 per 100 mL, the water should receive complete treatment consisting of coagulation-flocculation, sedimentation, filtration, and disinfection. Where the total coliform index exceeds 5 000 per 100 mL in more than 10 percent of the samples, auxiliary treatment consisting of pre-chlorination or pre-sedimentation, or their equivalents, and post-chlorination should be used. Other advanced forms of treatment approved by the control agency may be considered equivalent to any of the traditional methods named above.

2. If more than 10 percent of the raw water samples in any 30-day period have a fecal coliform density in the range 10 to 100 per 100 mL, or a total coliform density between 100 and 1 000 per 100 mL, the water should receive a combination of coagulation-flocculation, sedimentation, and filtration (partial treatment), or equivalent advanced forms of treatment approved by the control agency, followed by disinfection.

3. If any raw water sample contains fecal coliforms or if more than 5 percent of the samples in any consecutive 30-day period have a total coliform density greater than 10 per 100 mL, disinfection is required."

The World Health Organization (32) recommends that the raw water quality be such that given appropriate treatment equals or exceeds the guideline values for finished water.

Alberta Environment (33) requires a sanitary survey of a surface water source and treatment to produce water consistent with Canadian guidelines (31). The bacteriological objectives for surface waters used for potable water supply in Alberta are (34):

"1. In waters to be withdrawn for treatment and distribution as a potable supply or used for outdoor recreation other than direct contact, at least 90 percent of the samples (not less than five samples in any consecutive 30-day period) should have a total coliform density of less than 5,000 per 100 mL and a fecal coliform density of less than 1,000 per 100 mL.

2. In water used for direct contact recreation or vegetable crop irrigation the geometric mean of not less than five samples taken over not more than a 30-day period should not exceed 1,000 per 100 mL total coliforms, nor exceed these numbers in more than 20 percent of the samples examined during any month, nor exceed 2,400 per 100 mL total coliforms on any day."

The United States guidelines reviewed (35,40) recognized the importance of the fecal coliform test in assessing drinking water safety. This test was "preferred" since it is more specific for fecal contamination and not subject to wide range density fluctuation of doubtful sanitary significance (35). No source water criterion was established. The 1962 drinking water standards (37) also set no specific bacteriological criterion for water sources, simply stating that treatment should be sufficient to meet finished water standards. Interestingly, three other water quality documents between 1962 and 1976 (36,38,39) did specify bacteriological criteria for raw water sources for potable supply. These are summarized in Table 3.1.

3.2 The Significance of Current Guidelines/Standards

The various guidelines and standards reviewed reflect changes in interpretation of public health implications of the bacteriological raw water quality. Reliance on treatment processes to provide pathogen and indicator organism free product water for fair or poor raw water sources has been the result of increased faith in treatment technology. However, over the last 20 years treatment technology has changed little with regard to the basic treatment components. Furthermore data from the U.S. shows that waterborne

Table 3.1 Bacteriological raw water quality requirements (36,38,39).

Total Coliforms (no. per dL)	Fecal Coliforms (No. per dL)	Remarks	Reference
<10 000 ‡	<2 000 ‡	permissable criteria	(38)
<100 ‡	<20 ‡	desirable criteria	(38)
<100 ‡	<20 ‡	for waters requiring disinfection only	(36)
<20 000*	<4 000*	for waters using conventional treatment	(36)
<20 000*	<2 000*		(39)

*monthly geometric mean ‡ monthly arithmetic mean

disease outbreaks increased by a factor of five times between 1965 and 1980 (41). The number of outbreaks in 1980 was twice the number observed in 1946.

There is complete agreement amongst authorities that no coliforms should be present in finished drinking water (31,32,35). However, the U.S. EPA (35) stated that "in the monitoring of source water quality, fecal coliform measurements are preferred, being specific for fecal contamination and not subject to wide-range density fluctuation of doubtful sanitary significance." Health and Welfare Canada (31) agreed with the U.S. EPA and recommended the fecal coliform test as the most significant means for assessing fecal contamination of raw water sources. Neither agency specified the sampling frequency of raw water sources. IHD/WHO (42) suggested daily sampling of raw water sources for drinking water systems may be necessary. They also suggested the use of short-term surveys to assess the significance of short-term trends such as seasonal or diurnal variations.

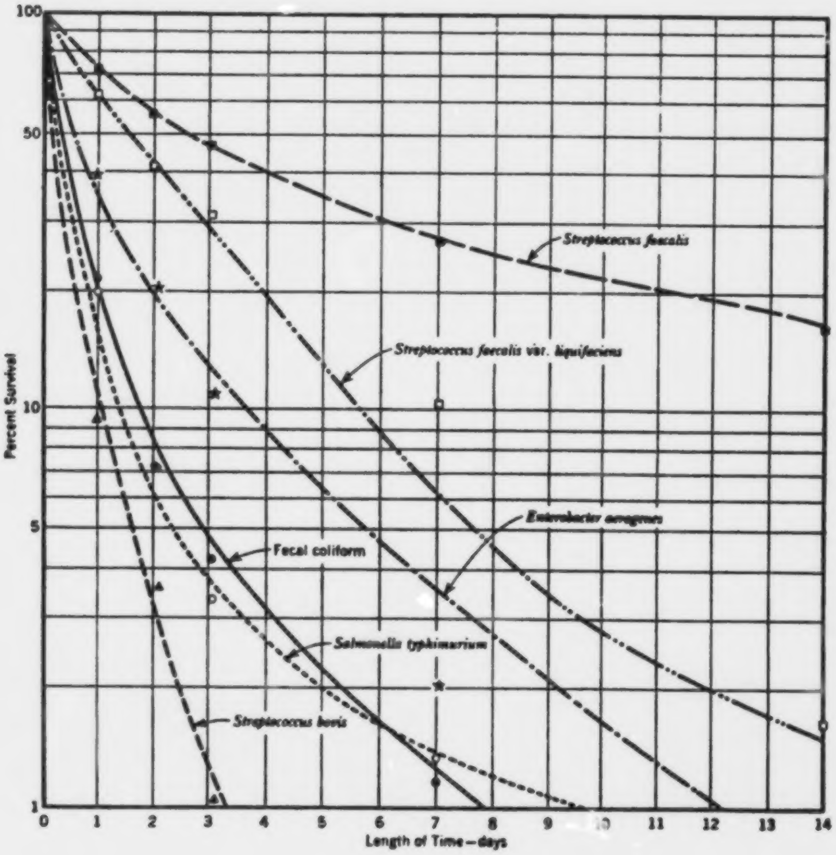
Geldreich and his co-workers discussed the sanitary significance of fecal coliforms and fecal streptococci compared to total coliforms in stream pollution in several articles in the late 1960's (43-47). They concluded that fecal coliforms were a much better indicator of fecal contamination than total coliforms and found the use of the fecal coliform/fecal streptococci (FC:FS) ratio informative under certain conditions.

Table 3.2 presents Geldreich's early data (43). He emphasized the care with which these ratios should be used since the ratio is sensitive to several parameters which include the type of sewage collection system, the chemical environment of the organisms, different die-off rates, and fecal streptococci biotypes which are not exclusively of fecal origin. Figure 3.1

Table 3.2 Typical fecal coliform and fecal streptococci densities from (42)

		Average indicator density per gram of feces		Average contribution per capita per 24 hr		
Animals	Average wt of feces/24 hr, wet wt, g	Fecal coliform, million	Fecal strepto-cocci million	Fecal coliform, million	Fecal strepto-cocci million	Ratio FC/FS
Man	150	13.0	3.0	2,000	450	4.4
Duck	336	33.0	54.0	11,000	18,000	0.6
Sheep	1,130	16.0	38.0	18,000	43,000	0.4
Chicken	182	1.3	3.4	240	620	0.4
Cow	23,600	0.23	1.3	5,400	31,000	0.2
Turkey	448	0.29	2.8	130	1,300	0.1
Pig	2,700	3.3	84.0	8,900	230,000	0.4

Figure 3.1 Persistence of some selected excreted bacteria in stormwater at 20°C. (47)



(47) illustrates the differences in die-off rate between the various biotypes of Group D streptococci and other organisms including fecal coliforms. Feachem et al. (48) reviewed the use of indicator organisms and the FC:FS ratio and concluded fecal streptococci had little value in sanitary surveys and the FC:FS ratio provided little reliable information about the origins of fecal contamination.

Feachem et al. (48) also reviewed the occurrence and survival of fecal indicators in surface waters. Stormwater was identified as being a major source of fecal pollution. Olivieri et al. (49) found Baltimore, MD. stormwater to have the characteristics of weak sewage. The density of fecal streptococci tends to be greater than fecal coliforms in separated storm sewer systems (43). Table 3.3 presents data collected by Geldreich, Kenner and others (45,46) for domestic wastewater and various types of stormwater. Table 3.4 shows summary fecal coliform data for various urban land use (50).

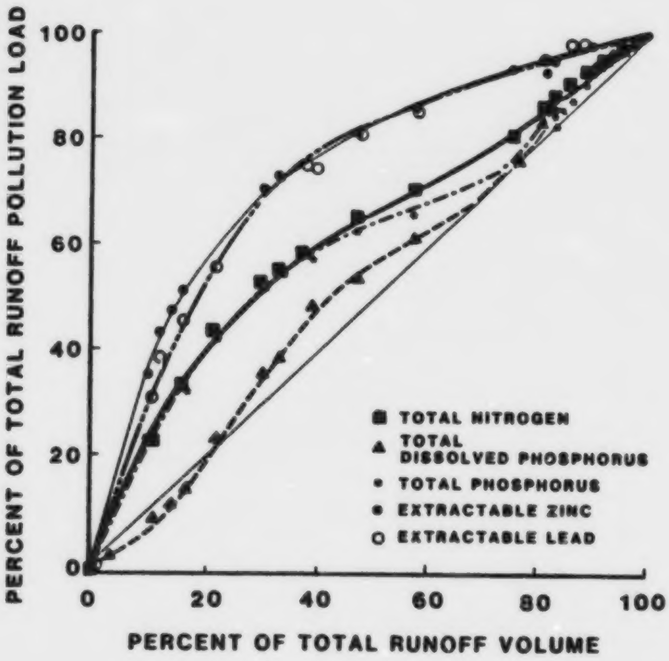


Figure 3.2 Runoff Pollution Timing Curves

The conclusion from the above discussion is that fecal coliform bacteria are the preferred indicator for assessing the sanitary quality of raw water sources. Total coliforms remain the preferred indicator for treated water. Fecal streptococci have limited value for assessing the sanitary quality of surface waters. They are particularly useful in differentiating storm drainage from sanitary sewage.

There has long been a misunderstanding regarding the use of indicator bacteria and their relation to microbial pathogens such as viruses, protozoa (particularly the encysted state), and helminth ova. The World Health Organization (51) reviewed the occurrence of viruses in water and concluded that bacterial indicators were unsuitable for indicating the potential presence of viruses since many pathogenic viruses persist better than the common indicator bacteria. Thus water which appears unpolluted, may actually have significant numbers of viruses. Similar conclusions have been reached for encysted protozoa and helminth ova (48).

It may be concluded from the foregoing discussion that fecal indicator bacteria may currently be the best means of quickly identifying polluted water. However their absence does not necessarily preclude the presence of pathogenic organisms. In this light, recommendations and practice elsewhere indicate that the fecal coliform test is the best bacteriological parameter for identifying potential public health problems in surface raw water sources. It is recommended that this parameter be used in Edmonton.

Table 3.3 Distribution of fecal coliforms and fecal streptococci in municipal wastewater (a) and stormwater (b). (45,46).

(a)

Water Source	Densities/100 ml*		Ratio FC/FS	Total Strains Examined	Occurrence (%)			
	Fecal Coliforms	Fecal Streptococci			Enterococci	*S. bovis* *S. equinus*	Atypical *S. faecalis*	*S. faecalis liquifaciens*
Domestic wastewater								
Preston, Ida.	340,000	64,000	5.3	39	79.5	None	None	20.5
Fargo, N. D.	1,300,000	290,000	4.5	50	100.0	None	None	None
Moorehead, Minn.	1,600,000	330,000	4.9	50	90.0	10.0	None	None
Cincinnati, Ohio	10,900,000	2,470,000	4.4	428	71.5	2.8	1.6	24.1
Lawrence, Mass.	17,900,000	4,500,000	4.0	50	84.0	4.0	None	12.0
Monroe, Mich.	19,200,000	700,000	27.9	70	78.6	1.4	4.3	15.7
Denver, Colo.	49,000,000	2,900,000	16.9	70	85.7	11.4	2.9	None
Stormwater								
Business district	13,000	51,000	0.26	1,476	78.5	1.6	1.2	18.8
Residential	6,500	150,000	0.04	1,158	80.0	0.5	1.4	18.1
Rural	2,700	58,000	0.05	445	87.4	0.5	0.2	11.9

* Median values.

(b)

Source	Date	Total Samples	Season	Total Coliform	Fecal Coliform	Fecal Streptococcus	Ratio FC/FS	Percent Fecal Coliform
Wooded hillside	Feb. 62 to Dec. 64	278						
			Spring	2,400	190	940	0.20	7.9
			Summer	79,000	1,900	27,000	0.70	2.4
			Autumn	180,000	430	13,000	0.03	0.2
			Winter	260	20	950	0.02	7.7
Street gutters	Jan. 62 to Jan. 64	177						
			Spring	1,400	230	3,100	0.07	16.4
			Summer	90,000	6,400	150,000	0.04	7.1
			Autumn	290,000	47,000	140,000	0.34	16.2
			Winter	1,600	50	2,200	0.02	3.1
Business district	Apr. 62 to Jul. 66	294						
			Spring	22,000	2,500	13,000	0.19	11.4
			Summer	172,000	13,000	51,000	0.26	7.6
			Autumn	190,000	40,000	56,000	0.71	21.1
			Winter	46,000	4,300	28,000	0.15	9.4
Rural	Jan. 63 to Aug. 64	94						
			Spring	4,400	55	3,600	0.02	1.3
			Summer	29,000	2,700	58,000	0.05	9.3
			Autumn	18,000	210	2,100	0.10	1.2
			Winter	58,000	9,000	790,000	0.01	15.5
Rainwater	Jun. 65 to Feb. 67	49						
			Spring	<1.0	<0.3	<1.0	—	—
			Summer	<1.0	<0.7	<1.0	—	—
			Autumn	<0.4	<0.4	<0.4	—	—
			Winter	<0.8	<0.5	<0.5	—	—

Table 3.4. Summary of Fecal Coliform Concentrations Observed During Study of Nonpoint Pollution Loadings (50).

Land Use	No. of Observations	Mean Concentration (MPN/dL)	Range (MPN/dL)	
			Minimum	Maximum
Large-lot single family residential (2.5-50 DU/ha)	19	1.5×10^4	2.8×10^3	1.5×10^5
Medium density single family residential (5.0-20 DU/ha)	25	1.17×10^4	---	0.43×10^5
Townhouse/garden apartment (20-54 DU/ha)	38	1.37×10^5	---	2.4×10^6
Suburban shopping center	41	1.01×10^5	---	2.4×10^6

DU = Dwelling Units

3.3 Evaluation of the North Saskatchewan River Data in Light of Prevailing and Possible Future Raw Water Quality Guidelines

The numbers and trends in the indicator bacteria densities upstream of Edmonton are insignificant from a public health point of view based on historical data examined (5-9). The highest densities of total coliforms were generally observed in June, July and August. Total coliforms have little sanitary significance since they can include soil organisms which are ubiquitous (48).

Examining Figure 2.2 for the six year period at E.L. Smith seasonal trends are evident. Peak coliform densities coincide with April/May suggesting spring break-up and runoff is significant. The occasional summer peak occurred and was probably associated with a period of wet weather. Fecal coliforms remained generally low. The occasions where the fecal coliforms exceeded 100 colonies per dL were in June or July of 1982, 1983, 1984. Generally the raw water quality was better than the guidelines for irrigation, contact recreation and water supply (31,40,52). The 95% confidence intervals for the geometric mean of total and fecal coliforms indicates the high degree of reliability for the E.L. Smith raw water meeting bacteriological criteria.

Microbial water quality at the Rossdale Water Treatment Plant intake poses a much more complex problem as a result of urban pollution in the reach from E.L. Smith to Rossdale. Table 1.1 and Figure 2.3 illustrates the complexity. Again spring break-up and summer storm events tend to produce noticeable peaks. However, there is a significant background level of fecal contamination throughout the year. This is illustrated by the 95% confidence interval for the geometric mean fecal coliform density. The geometric mean is highly likely to exceed the guidelines for recreational activities (52). The current treatment processes at Rossdale satisfy the existing requirements described by Health and Welfare Canada (30) for potable water sources where conventional treatment is provided.

Current microbiological guidelines for raw water supplies are somewhat lax with respect to organisms which have been known to penetrate water treatment plants similar in design and complexity to E.L. Smith and Rossdale. Viruses and encysted protozoa have been known to penetrate multiple barriers when processes are poorly operated (48,53,54). Until better virus and protozoa cyst enumeration techniques and/or indicators are developed, indicator bacteria need to be used cautiously. Some investigators found appreciable attack rates for gastroenteritis in swimmers who bathed in water with very low Streptococcus faecalis and Escherichia coli densities (55).

In conclusion, fecal pollution in a raw water supply poses challenges to a water treatment plant. There is a risk that through unforseen major pollution episodes or human error, the treatment may be breached by pathogenic microorganisms and owing to the 1 or 2 day tests required for indicator bacteria, it would be too late for remedial action. Therefore, if a good quality, unpolluted source is available, this should be the location for potable water supply to reduce the risk of waterborne disease.

3.4 Comparisons With Other Communities

There are as many challenging upstream water quality situations as there are major water users. Each location has features which require special consideration during design and operation of the water treatment plant. However, a number of communities have found one or more of their water treatment plants assaulted by unwanted contamination. The approaches used to deal with such assaults are the subject of this section.

3.4.1 Britannia Water Purification Plant, Ottawa, Ontario

The construction of this plant was initiated in 1959 after careful review of many years of water quality data from the Lemieux Island Water Treatment Plant which takes water from mid-channel in the Ottawa River (56). Initial operation of the plant was challenged by taste and odour problems as well as elevated chlorides and conductivity. Modification of an upstream island diverted some mainstream water past the area of the water intake resulting in improved raw water quality (see Figure 3.3). By 1972 poor water quality resulted in the initiation of a major four year water sampling program. It was found that a small creek located downstream of the water intake was the major source of contamination. Urbanization along the creek resulted in greatly increased microbial and organic contamination.

The sampling program relied on fecal coliforms as the indicator of raw water contamination. A value of 100 fecal coliforms per dL was selected as the dividing point between contaminated and non-contaminated water. Daily samples at the original intake (which is routine) and at a number of extension locations over several years confirmed the need for a new water intake located well into the main channel of the Ottawa River to assure raw water of the highest possible quality for treatment. A frequency of fecal coliform values over 100 per dL versus recurrence interval was used to select the extension (see Figure 3.4). It was concluded by those involved in the development of the new water intake that "raw water quality for a particular source of supply should rank in importance with the treatment process and distribution network in the development of a municipal water system."

The major similarity between this plant and the Rossdale plant is that they both deal with a river raw water source which was subject to storm and break-up caused pollution assaults. In both cases taste and odour, and microorganism concentrations were of concern. Changes to the intake location at the Britannia Water Purification Plant came after a careful study of the raw water fecal coliform quality available at different locations.

3.4.2 Cincinnati Water Works, Ohio

Cincinnati's California Water Treatment Plant processes up to 890 ML per day. The original design recognized the high degree of pollution in the Little Miami River and recommended the water intake on the Ohio River. The raw water is pumped from the river to a chemical treatment building for coagulation and pre-settling. Depending on water quality and demand the water can be directed to outdoor settling reservoirs (1.5 x 10^6 m^3 capacity) or to coagulation basins for additional chemical addition. The unique component of

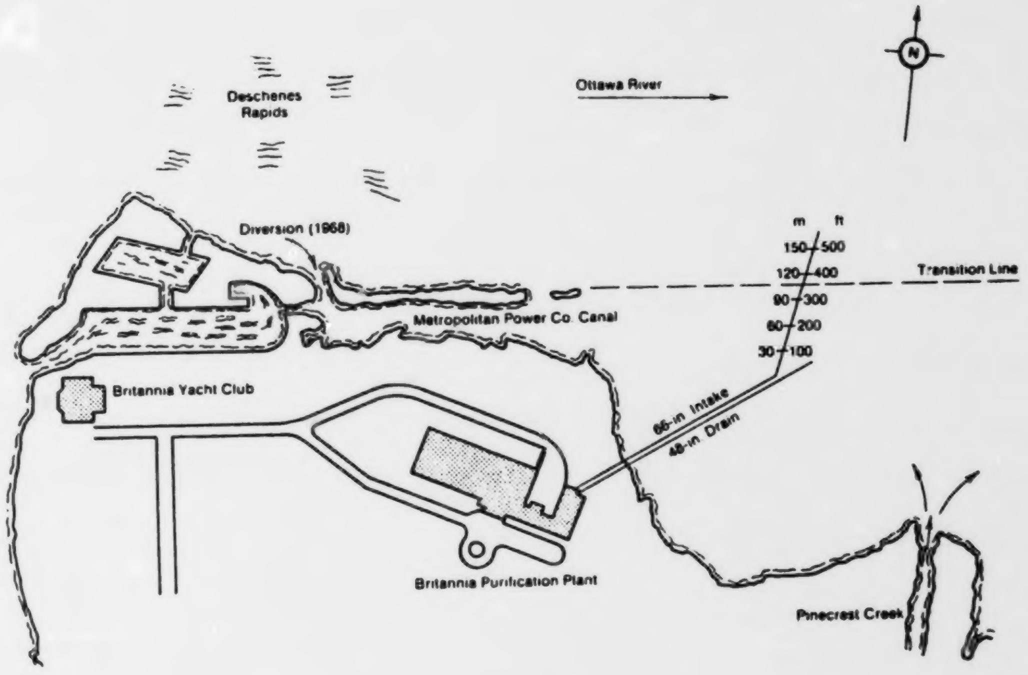


Figure 3.3 Area Plan of Britannia Water Purification Plant (54)

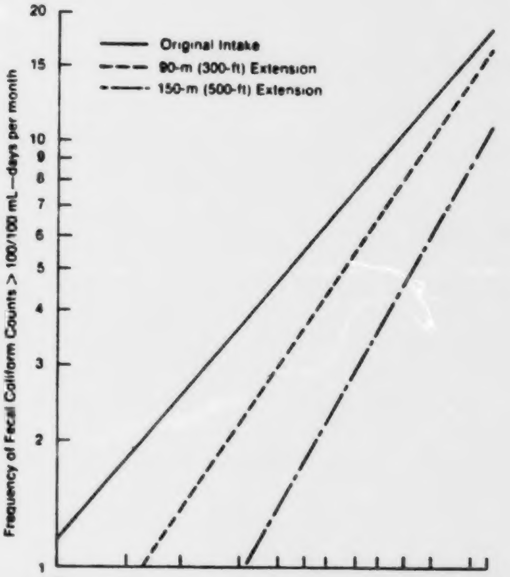


Figure 3.4 Frequency of recurrence of unacceptable fecal coliform counts for three intake locations (54)

the system is the settling reservoirs which allow a good opportunity for natural sunlight induced disinfection of the water. Figure 3.5 shows a diagram of this site.

This plant has a somewhat different operating procedure from that followed at Edmonton. The important factors are the water intake was located to avoid industrial and urban contamination, and they made use of large holding ponds to buffer their system, allow temporary suspension of withdrawals from the river if a contaminate was detected in the raw water, allow natural sunlight conditions to assist with disinfection, and to improve water quality through long-term settling.

4 Mixing Analyses

The river water quality at the Rossdale water intake is largely a function of the pollutants discharged from the sewer outfalls upstream of the intake. To determine this concentration, estimates of the configurations of the effluent plumes downstream of the various sewer outfalls upstream of the intake section are required.

4.1 Procedures and Assumptions

As there are 85 outfalls above the intake section and within the City limits, it is unreasonable to separately calculate the plume generated by the effluent from each outfall. Instead calculations were made for only a limited number of outfall locations between the Millwoods storm drain outfall and the intake section. The Whitemud Creek contribution was limited to the contribution of the outfalls. These calculated plumes were then used to define the variation in concentration caused at the intake by outfalls on both banks at different distances upstream. The curves showing this variation

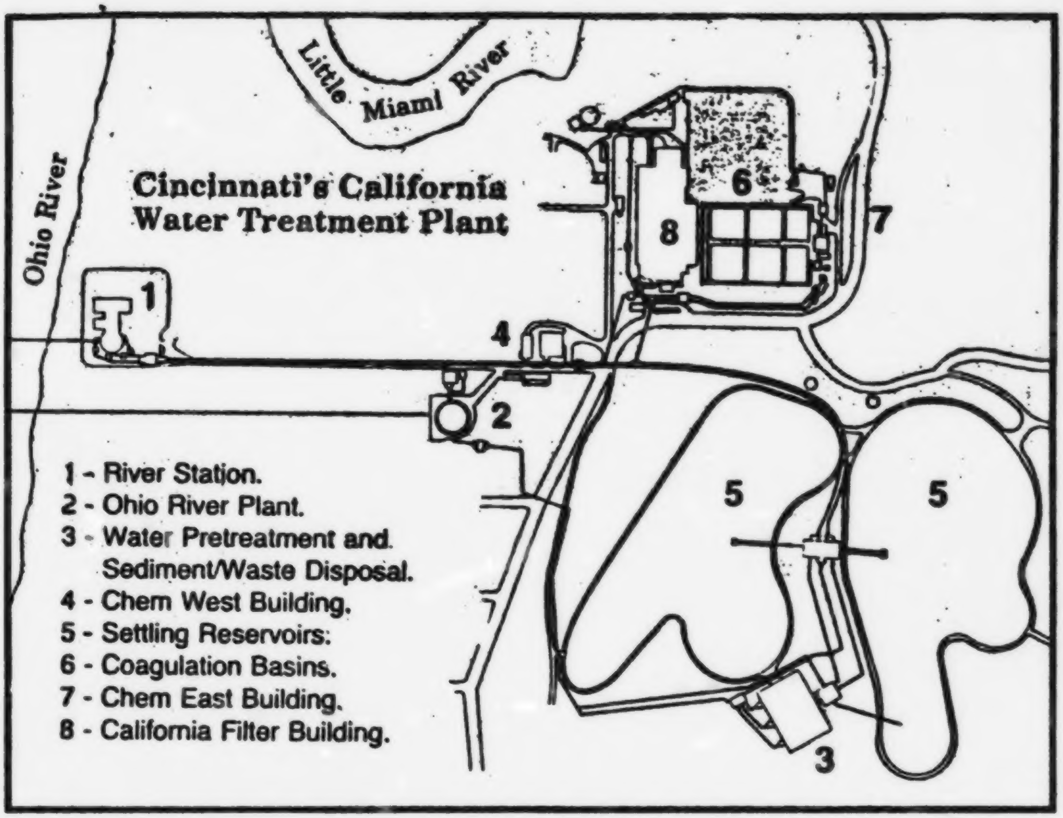


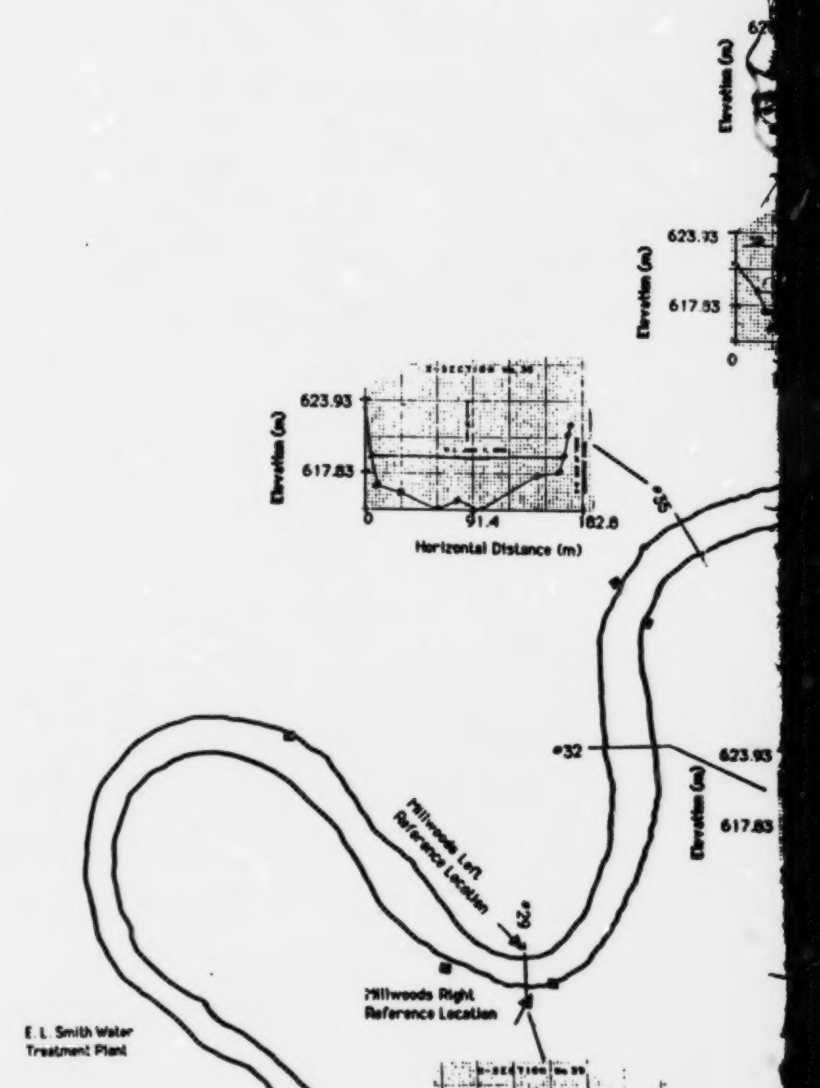
Figure 3.4 Diagram of Cincinnati's California Water Treatment Plant

result from a linear equation describing the plume. The concentrations at the intake due to various upstream outfalls can be determined directly from the 'influence lines'. The values can be added to find the total concentration at the intake due to all the upstream outfalls. This procedure is explained in detail in Section 4.3

The fundamental need is for an algorithm with which to calculate the plume characteristics for a given outfall location. Repeated application of this algorithm for the selected outfall locations would then permit the construction of the influence lines. The algorithm chosen was one that assumes that the pollutant discharge is steady (ie. constant in time) and, at any point in the river, is uniformly mixed over the depth (ie. no concentration variation over a vertical) but not across the channel. The latter assumption is standard for assessing concentrations at distances more than about 100 depths from the outfall [see, for example, Beltaos (57)]. The former is a simplifying assumption: if the pollutant discharge does vary substantially with time the analysis is much more difficult. The steady state assumption would seem adequate for the present study. Details of the algorithm used are given by Putz (58).

The algorithm's success depends largely on good definition of the hydraulic geometry of the reach of interest and selection of an adequate mixing coefficient for the reach, the latter being the measure of the ability of the turbulence in the flow to diffuse the pollutant across the channel as it moves downstream. For the present study the hydraulic geometry was represented by some selected river cross-sections over the reach of interest. These were provided by River Engineering Branch, Alberta Environment. The section locations are shown in Figure 4.1. An example is shown in Figure 4.2. The mixing coefficient selected was based on values reported by Beltaos and Anderson (59) for the reach downstream of Edmonton, and values given for other Alberta rivers by Beltaos (57). The water level at each cross-section, and hence the waterway geometry, depends on the discharge. The discharge selected for this study was 150 m^3/s. This was thought to be a fair estimate of a low spring discharge. For comparison, the highest minimum winter discharge since the Big Horn Dam was commissioned as 130 m^3/s. The outfalls were assumed to be 'point' sources of pollutant, with discharges that are small relative to that in the river.

Calculations of plume characteristics were carried out for the above 'datum' situation for outfalls on both banks at four locations: Millwoods outfall, Quesnell Bridge, Groat Road and the High Level Bridge. To



623.93
617.83
Elevation (m)
0
91.4
182.8
Horizontal Distance (m)
•32
Millwoods Left Reference Location
Millwoods Right Reference Location
E. L. Smith Water Treatment Plant

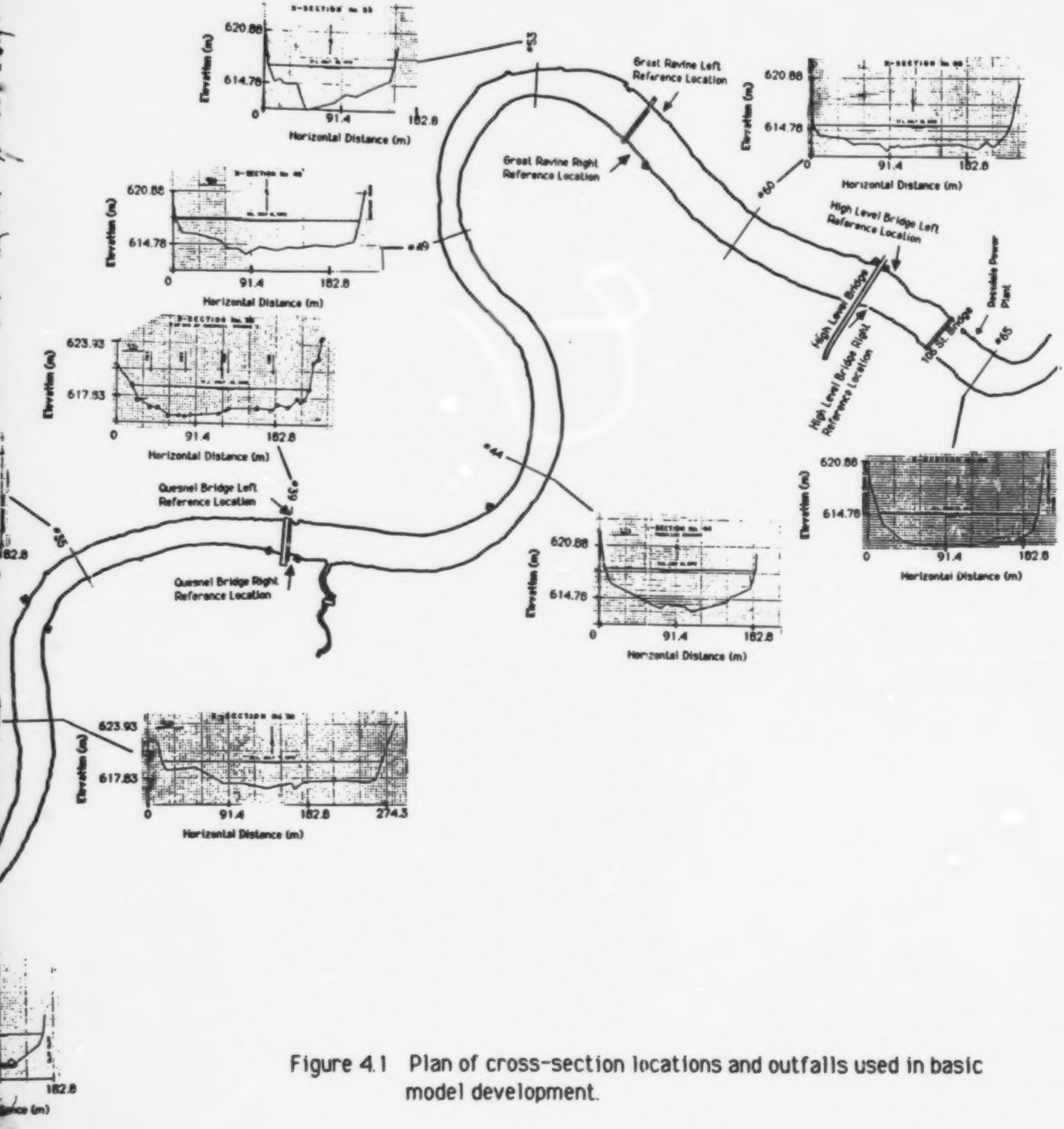

#53
620.88
614.78
0
91.4
182.8
Elevation (m)
Horizontal Distance (m)
Groat Ravine Left Reference Location
Groat Ravine Right Reference Location
#60
High Level Bridge Left Reference Location
High Level Bridge
High Level Bridge Right Reference Location
105 St. Bridge
Rossdale Power Plant
#65
#49
623.93
617.83
#44
Quesnel Bridge Left Reference Location
Quesnel Bridge Right Reference Location
#39
#35
274.3

Figure 4.1 Plan of cross-section locations and outfalls used in basic model development.

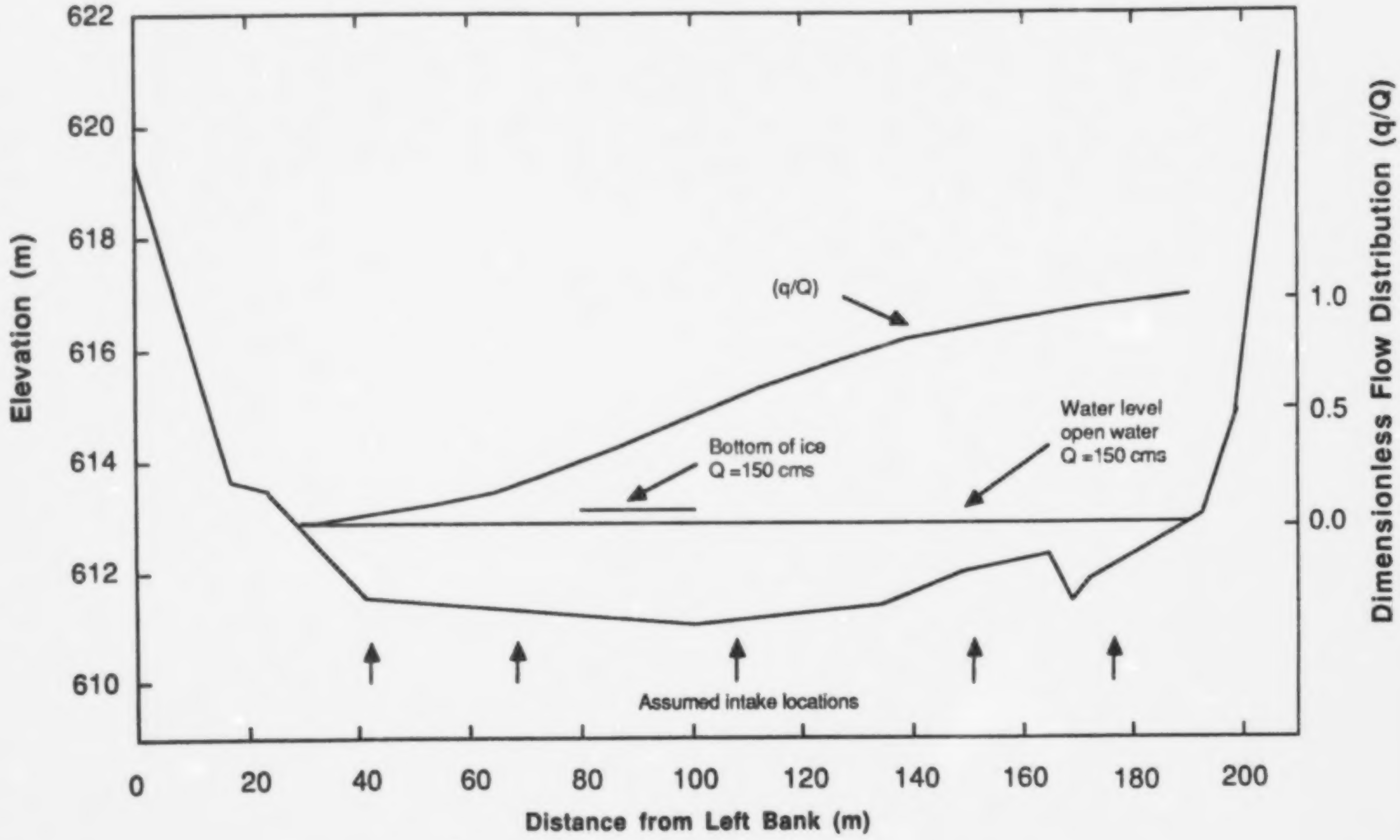


Figure 4.2 Example of dimensionless flow distribution at a cross-section

assess the sensitivity of the results to variations from this datum circumstance, changes in the plume originating at the Millwoods outfall were calculated for each of the following variations: ice-covered channel; river discharge doubled; mixing coefficient halved; and the outfall represented as a line source extending to 40 m from the bank rather than as a point source 20 m from the bank.

4.2 Calculated Plume Characteristics

The concentration variation across each of the plumes at the intake section from the eight 'reference' outfall locations are shown in Figure 4.3. The concentrations given in Figure 4.3 are relative to the fully-mixed concentration, C_∞, the concentration that exists when the pollutant discharge is uniformly mixed across the whole channel. It is given by:

$$C_\infty = \frac{\overset{o}{m}}{Q}$$

where $\overset{o}{m}$ is the rate of inflow of pollutant and Q the total discharge in the river. Such a concentration can be expected to exist across the channel beyond some 400 river widths downstream of the outfall (approximately 60 km).

It is evident from Figure 4.3 that the pollutants are far from fully mixed across the river at the water intake, even for outfalls as far upstream as Millwoods. To more directly indicate the plume width growth, Figure 4.4 shows the variation of plume width with distance downstream of the Millwoods outfall, the edge of the plume at any section being taken as the location where the concentration is 5% of the peak concentration at the bank at the section. (In Figure 4.4 the width has been given in 'discharge' coordinates: q represents the discharge between the left bank and any point across the section. This parameter is a more convenient and meaningful representation of transverse location than the direct distance from the

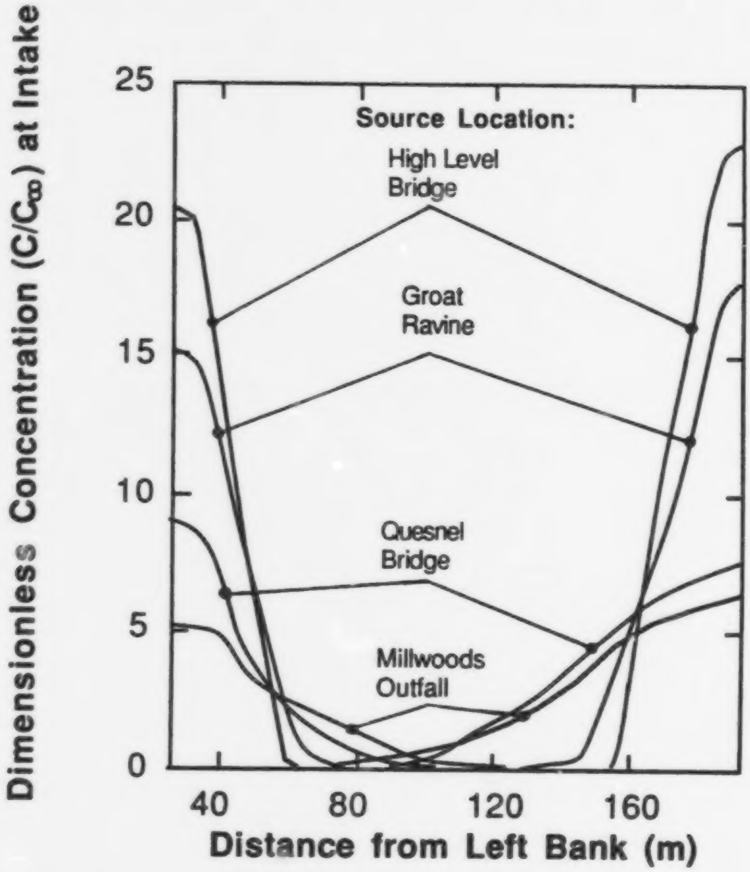


Figure 4.3 Effect of upstream right and left bank discharges on contaminate concentration at the Rossdale Water intake.

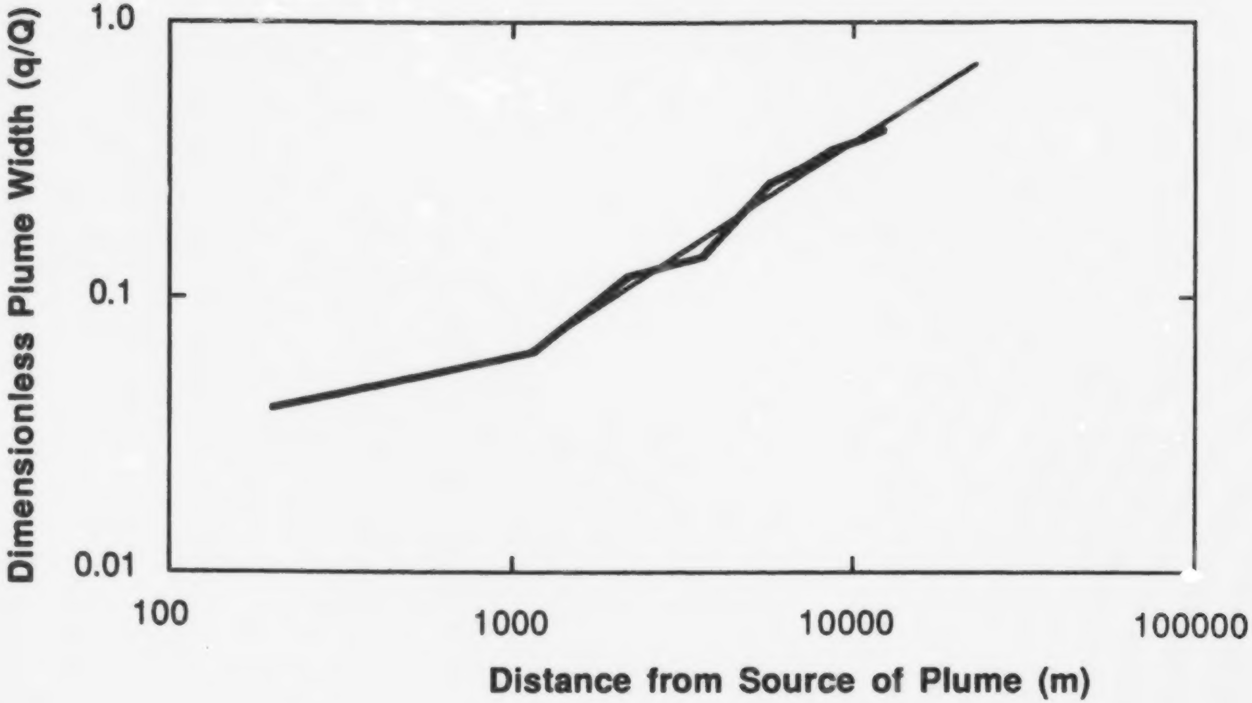


Figure 4.4 Variation in plume with downstream of outfall for reference situation.

bank. A typical variation of q with z is that at the intake section shown in Figure 4.2. It will be noted that, by definition, q must vary from zero at the left bank to Q at the right bank, no matter what the actual water surface width is.)

Concentration influence lines for an intake near the left bank, at 1/4 of the water surface width from the left bank, at the water surface centre, at 3/4 of the water surface width, and near the right bank, are shown in Figures 4.5(a), for outfalls on the left bank, and 4.5(b), for outfalls on the right bank. Evidently the influence of an outfall on the concentration at an intake near a bank decreases substantially as the distance between outfall and intake increases. However, for an intake well out in the stream the influence of an outfall at a bank first increases with distance then decreases. This is due to the combined effects of the concentration decrease downstream of an outfall and the plume width increase. Indeed, for the 'datum' circumstance on which Figures 4.5(a) and (b) are based, an intake to the right of centre stream would be more-or-less free of the influence of pollutants from outfalls on the left bank downstream of Millwoods although, as Figure 4.5(b) shows, it could then be within the influence of outfalls on the right bank.

Figure 4.6 is intended to illustrate the sensitivity of the above 'datum' solution to changes in important parameters. The figure shows changes in the calculated concentration distribution at the intake section, caused by the different values of the parameters indicated in the figure, for an outfall at the location of the Millwoods outfall. Evidently the distribution is not sensitive to the assumed outfall configuration, there being little difference between assuming the outfall is a point source located 10 m from the bank and a line source that extends out 20 m from the bank. However the distribution

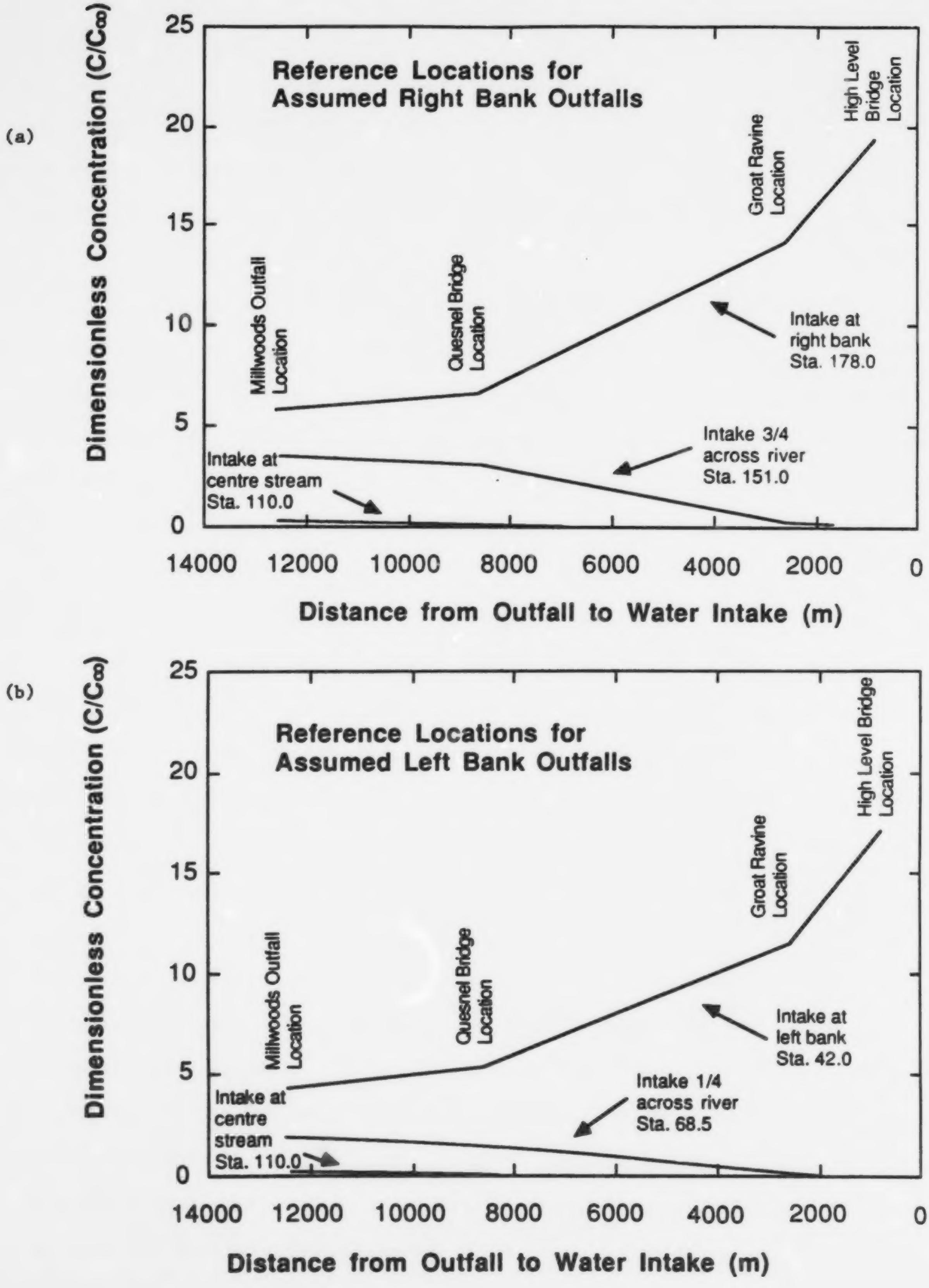


Figure 4.5 Effect of left (a) or right (b) bank discharges on the dimensionless

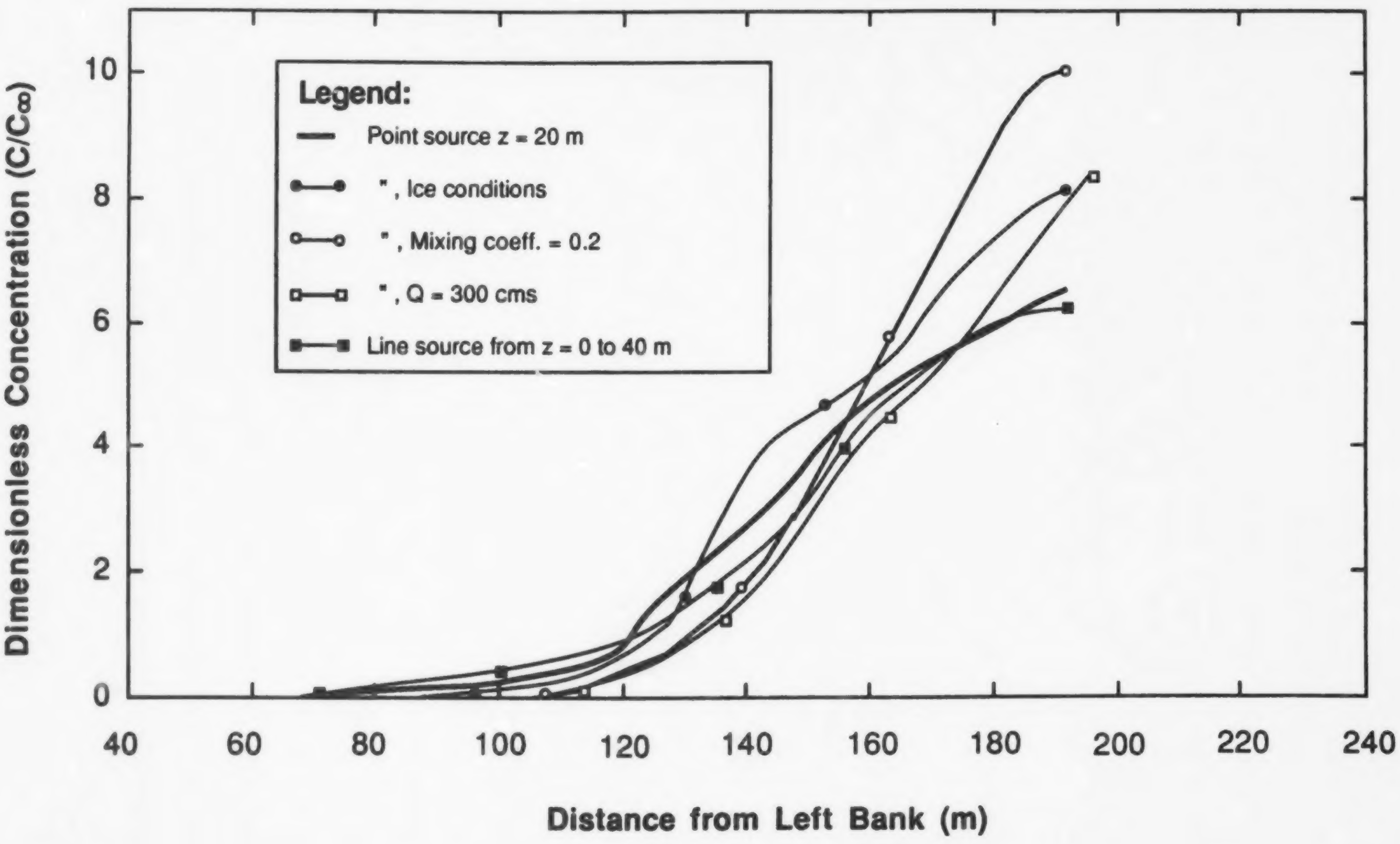


Figure 4.6 Impact of various parameter values on the Millwoods plume at the Rossdale intake.

is sensitive to changes in the assumed river discharge and mixing coefficient, and to whether there is an ice cover or not. An ice cover, an increase in discharge and a decrease in mixing coefficient all cause an increase in concentration near the bank but a decrease in plume width at the intake section. This plume width variation is also shown in Figure 4.7, where here the plume width has been taken to a point of constant concentration, where $C/C_\infty = 0.25$, instead of 5% of the concentration at the bank used in Figure 4.4, which gives a variable concentration.

4.3 Example Scenario

Using the information presented in Section 4.2 an infinite number of discharge and contaminate concentration scenarios can be developed for the purpose of estimating the impact on water quality at the Rossdale intake. For demonstration of the procedure the 85 upstream outfalls have been assembled into eight point discharge groups. Using Figures 4.5(b) for the right bank discharges and Figure 4.5(a) for the left bank discharges the contribution of fecal coliform by each outfall above the background numbers can be estimated. The details of the scenario development are provided in Appendix B. The major components of the scenario are provided in Table 4.1.

The selection of 0.25 of the 5 year storm flow was based on the best available information from the City of Edmonton, Water and Sanitation Department. The estimated 5 year storm flow information had been developed recently in order to establish a better understanding of flow to the North Saskatchewan River between the E.L. Smith and Rossdale plants (3). The flow estimates for the storm sewers were developed from a graph of flow versus diameter for outfalls with known discharges.

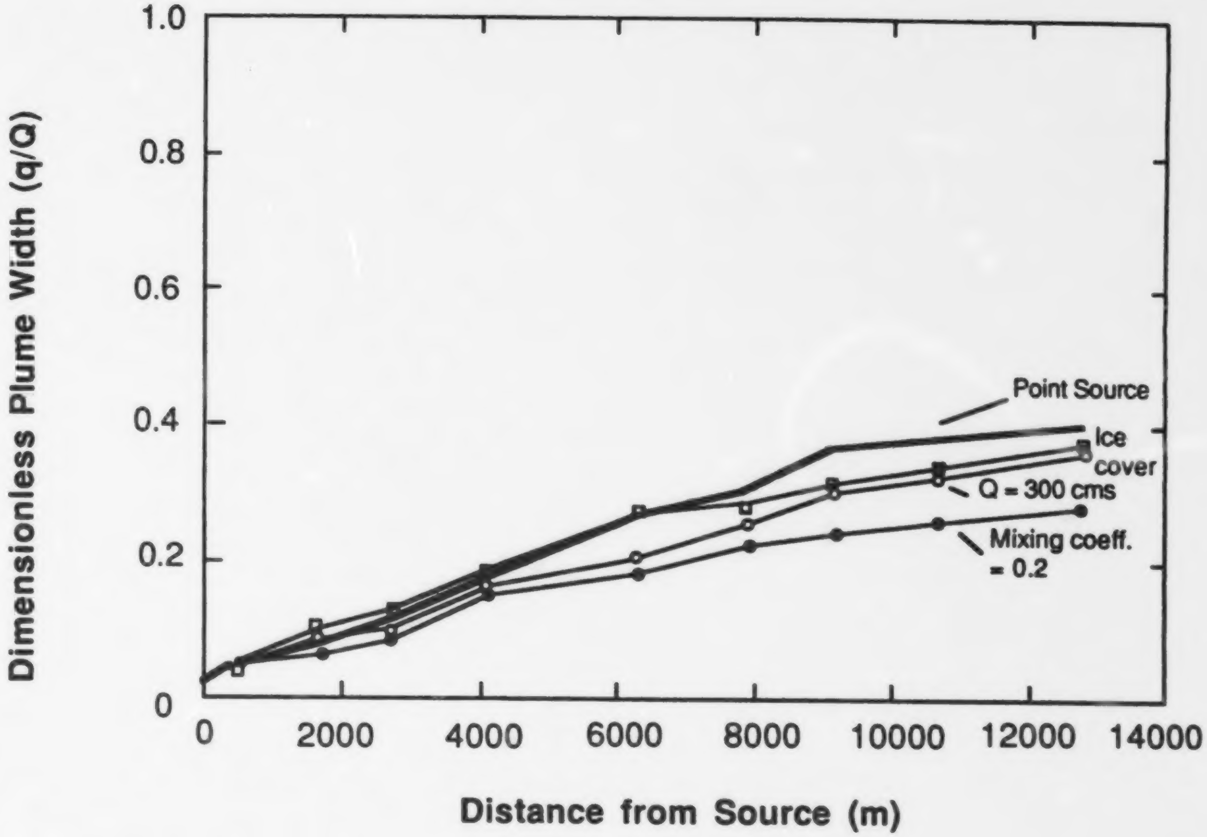


Figure 4.7 Variation of plume width for variation in assumed parameters.

TABLE 4.1

Major Components of Example Scenario

	Component	Value
A.	Number of Outfall Groupings	7
B.	Flow etimate for outfalls with overflow from sanitary and combined sewers	(0.25) (5y storm flow)
C.	Flow estimate from combined sewers	0.5[(0.25) (5y storm flow)] + 0.5[Normal sewer flow]
D.	Flow estimate for storm sewers	Graphical estimate (0.25) (5y storm flow)
E.	Background fecal coliform concentration	10 FC/dL
F.	Wastewater fecal coliform concentration	2×10^6 FC/dL
G.	Storm water fecal coliform concentration	5×10^4 FC/dL
H.	Die-off rate in river	0

Fecal coliform concentrations for various types of discharges were based on actual sampling results from Edmonton locations and literature values. Wastewater contributions were estimated from data collected from the influent to the Gold Bar Sewage Treatment Plant over the last few years. Storm water concentrations were established after review of the 1986 Groat Road Outfall (32) data and literature values reported earlier.

The individual impact of the group discharges to the river are shown in Figure 4.8. Figure 4.9 illustrates on a map of the North Saskatchewan River the approximate area affected by the plume from outfall groups R-I (Millwoods outfall area), L-II (Quesnel Bridge area) and R-III (Groat Road area).

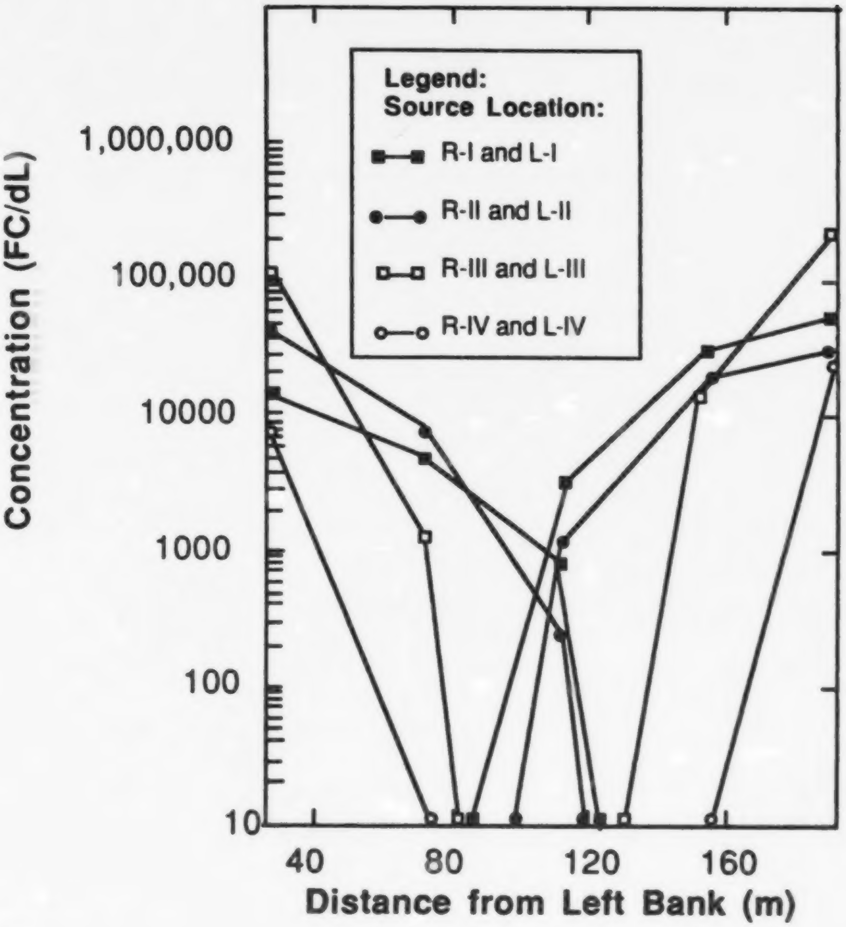


Figure 4.8 Concentration of fecal coliform at the Rossdale cross-section for eight upstream outfall groups.

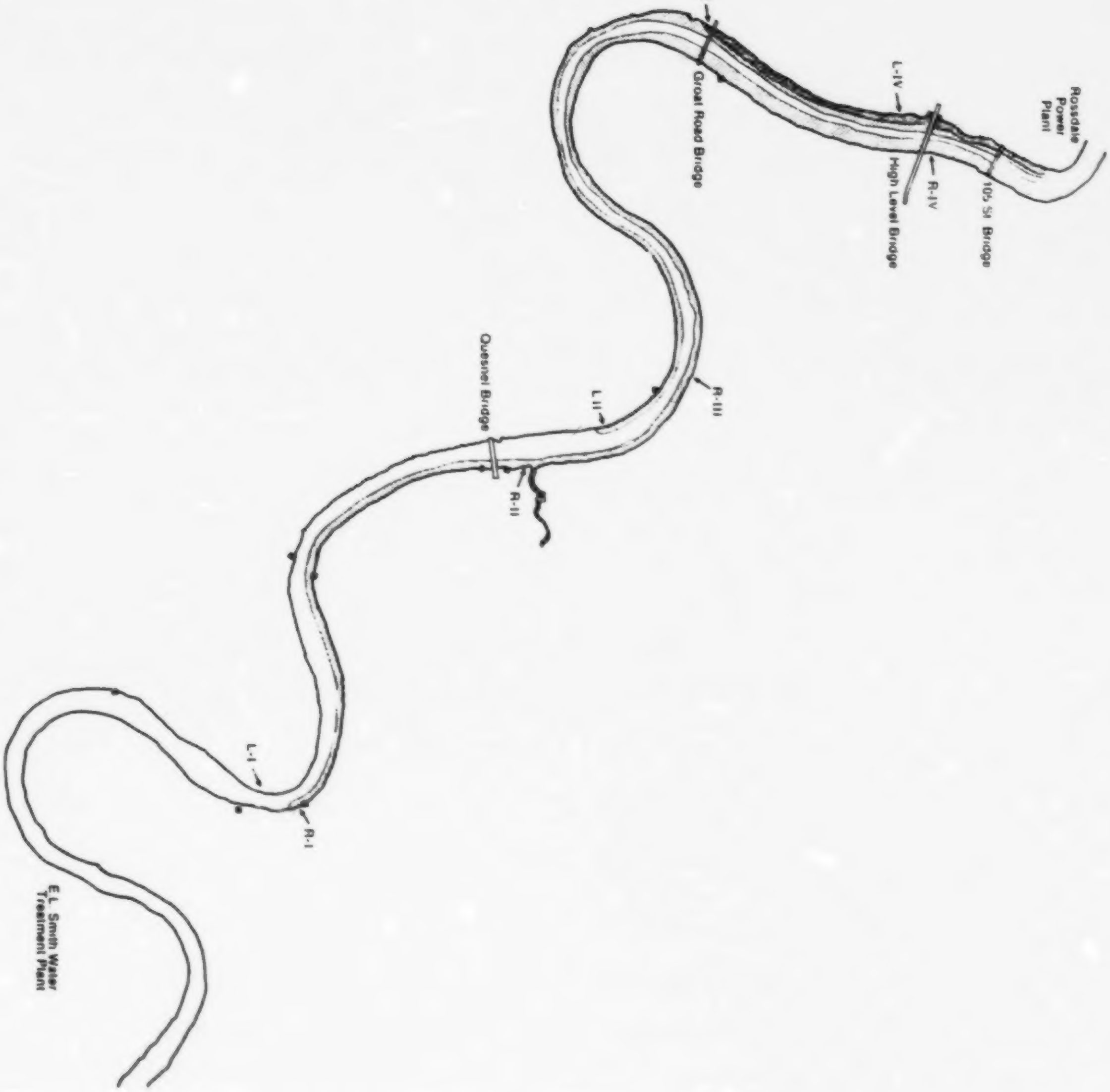


Figure 4.9

Area of North Saskatchewan River affected by three different outfalls.

4.4 Implications for Raw Water Intake

The scenario provided is only one of many which could be examined. It has been developed as a conservative model of what could occur. The numbers used were based on the best available data. However a much more detailed and comprehensive study with field varification is required.

Figure 4.10 presents the summation of the curves in Figure 4.8. What is indicated is that the concentration of fecal coliform at the banks and the centre of the North Saskatchewan River could reach values well in excess of the numbers recommended for surface water supplies. This situation is in contradiction with the basic concept of water supply quality assurance through the use of multiple barriers.

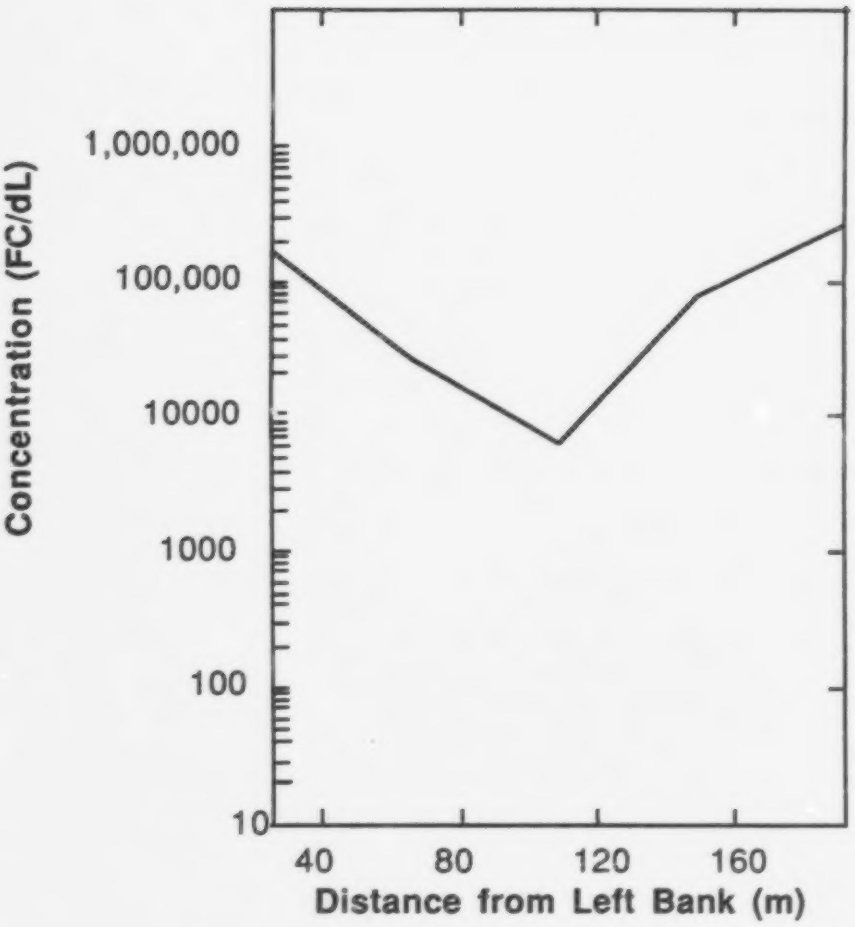


Figure 4.10 Summary concentration diagram for the Rossdale cross-section.

4.5 Impact of the Chlorinated Discharge at Devon

The wastewater treated at Devon is disinfected with chlorine prior to release. A quick look of characteristic mixing patterns in the North Saskatchewan River suggest that the bank discharge may have moved out about 40% of the river width by the time it reaches the E.L. Smith intake cross-section. See Figure 4.7.

5 Mitigation Options

The microbiological quality of the raw water at the Rossdale plant consistently exceeds a value to 100 FC/dL. The mean value for the last six years is 166 FC/dL. The example scenario developed suggests that storm events may cause considerably higher values at all points along the cross-section. Two categories of mitigation techniques are considered here: leaving the intake at its present cross-section and moving it upstream.

5.1 Intake Location at Present Cross-Section

Leaving the raw water intake at the present location appears to be at odds with the good practice of assuring the best possible quality of raw water before applying treatment. However there are some options which could be followed to improve the raw water quality. These are (a) significantly improve treatment capabilities, (b) improve raw water quality by reducing contamination upstream, and (c) adjusting the location of the intake in the cross-section.

5.1.1 Improved Treatment Capabilities

The science of destruction of microorganisms in water has improved significantly in the last 15 years. The effectiveness of chlorine, chloramines, chlorine dioxide and ozone have been studied to varying degrees. Below are some general conclusions which can be drawn about these disinfectants.

1. Ozone is the best and the most expensive agent for the destruction of viruses, bacteria, protozans and helminths. It does not provide a protective residual in the distribution system.
2. Chlorine dioxide is a strong disinfectant. It can result in higher levels of chlorite in the product water which may be of concern.
3. Chlorine is the best known and most widely used disinfectant in North America. It is not as effective against the range of microorganisms as the above disinfectants.
4. Chloramines is by far the poorest of the disinfectants listed. It also may be the cheapest to use.

The current use of some chlorine dioxide supplemented by chloramines for final disinfection and distribution system protection would need re-evaluation with this mitigation action.

In plant control of coagulation, flocculation, sedimentation and filtration would require frequent and careful control. More redundancy of equipment to insure performance of the plant would be necessary.

5.1.2 Improvement of Raw Water Quality

The 85 outfalls between the E.L. Smith plant and the Rossdale plant have a significant impact on the microbiological quality of the raw water. The Water and Sanitation Department has addressed the issue of clean-up discharges to the North Saskatchewan River. The seven main combined sewer overflows upstream of Rossdale are scheduled for elimination by 1987. In addition there is an attempt to remove sanitary sewage overflow connections to storm sewers which are located upstream of the water intake as soon as possible. A major portion of this work is scheduled to be completed in 1986.

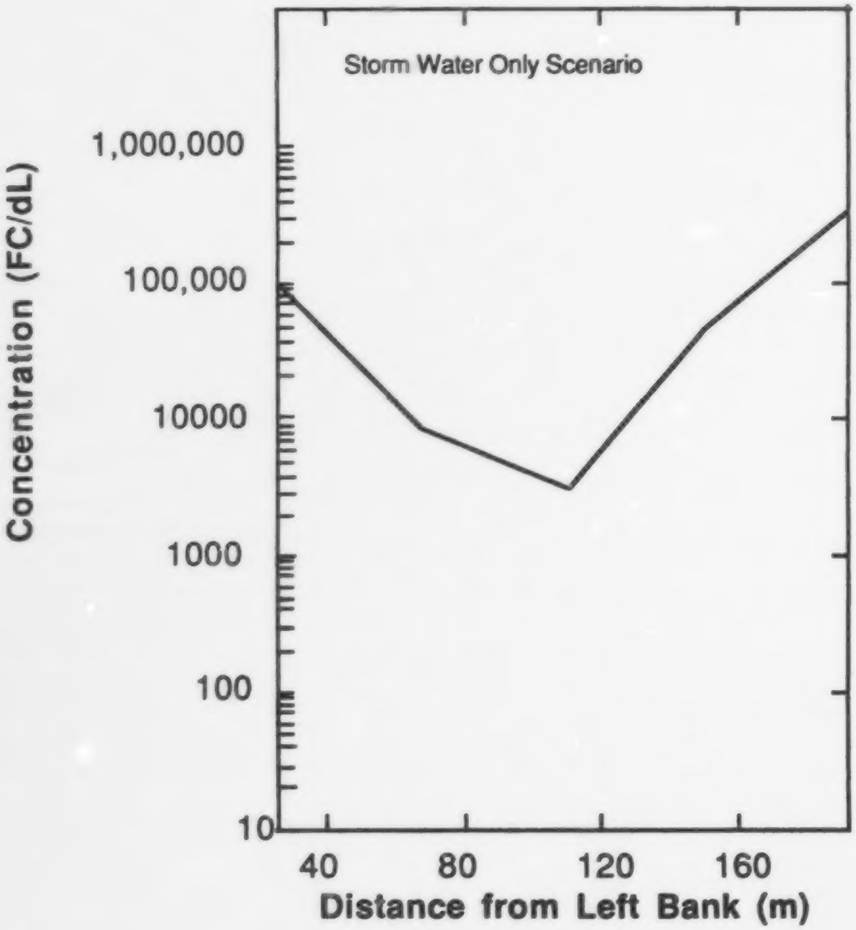


Figure 5.1
Example Scenario Assuming No Combined or Sanitary Sewer Discharges

The above activities will have a significant impact on the results of the Example Scenario. Figure 5.1 shows the results of the Example Scenario with no combined or sanitary sewer discharges.

5.1.3 Adjusting Location of Intake in Cross-Section

Both Figure 4.3 and 5.1 show that significant improvement in water quality can be expected by moving the raw water intake to the middle of the river. It should be noted that the fecal coliform quality for a major pollution event like that in the scenario are still very high.

5.2 Intake Relocation Up-Stream

The relocation of the raw water intake has several important benefits which make this alternative interesting. First, total elimination of combined sewer overflows and sanitary sewage discharges upstream of the intake, although reducing the total amount of contamination, will not eliminate microorganisms in the raw water. As shown earlier storm water is rich in fecal coliform microorganisms. Furthermore, animal origin microorganisms may, on occasion, be a very significant component of the population. Since some animals can be an intermediate host for microorganisms like Giardia, there is still concern about improvement of raw water quality.

Moving the Rossdale water intake upstream could result in varying degrees of raw water quality improvement. For example, moving the intake to a location about 5000 m upstream of the Rossdale plant (about 2200 m upstream of the Groat Road Bridge) would produce a much different looking concentration distribution. This is due to the elimination of about 47 outfalls from the contributing to the raw water quality. This is shown in relative terms through the use of the Example Scenario conditions (see Figure 5.2).

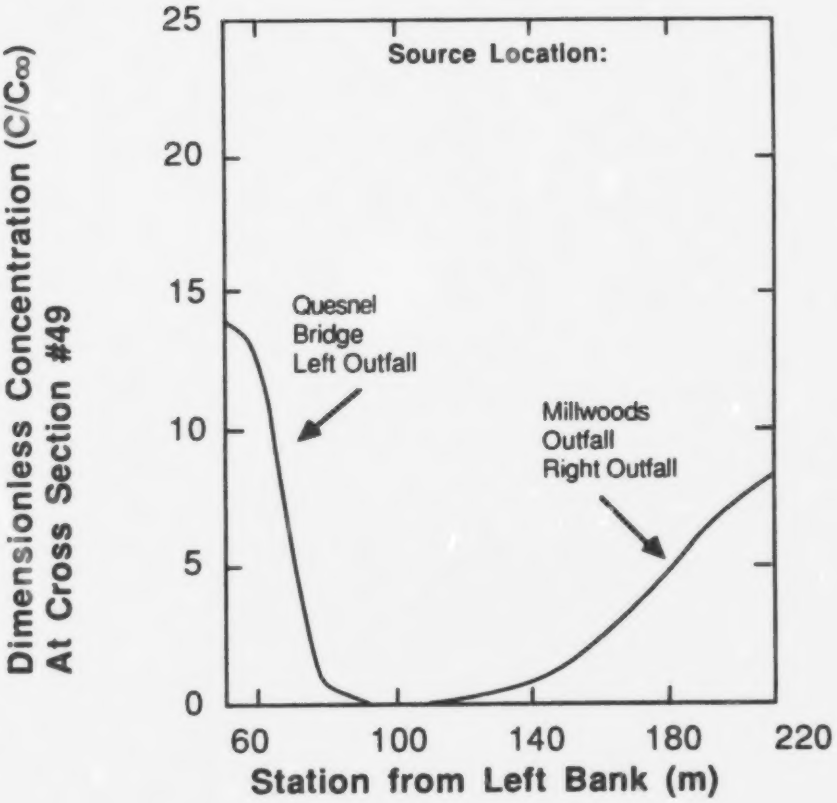


Figure 5.2 Plume distribution at a cross-section approximately 2200 m upstream of Groat Road Bridge (No. 49).

By moving the intake upstream another 2000 m the Example Scenario conditions would be as shown in Figure 5.3. This figure suggests that the impact of the two major outfalls would remain well away from a properly located centre of river intake. This location could be reached by constructing a tunnel about 3400 m in length under Garneau, the University of Alberta, and the southern edge of Windsor Park.

Since both sites are still subject to contamination through new upstream outfalls related to City growth, and road and bridge spills of contaminates, the ultimate solution would be combining the water intakes for E.L. Smith and Rossdale at the E.L. Smith site.

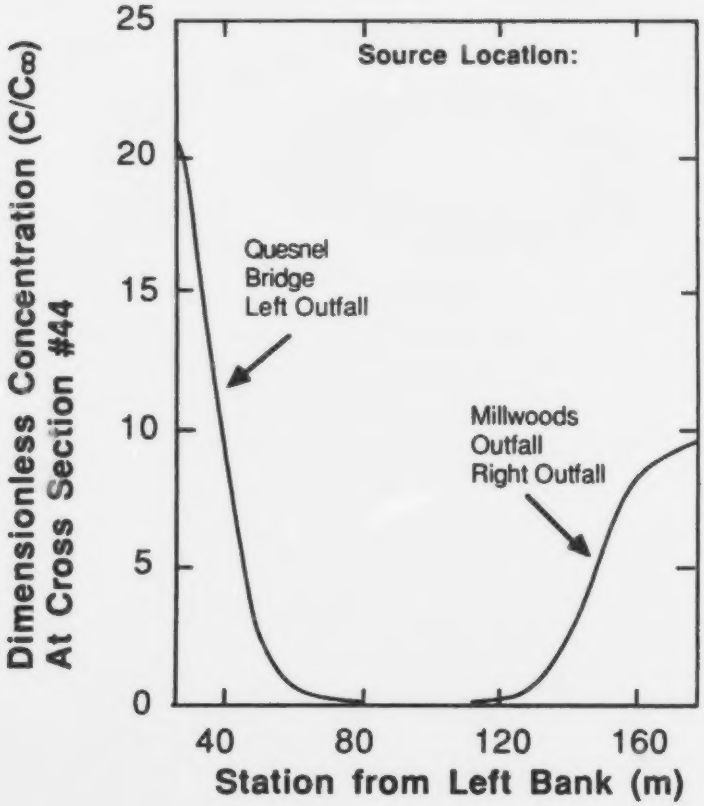


Figure 5.3 Plume distribution for cross-section 44 west of University of Alberta

6 Conclusions and Recommendations

The conclusions and recommendations presented here only relate to the aspects of raw water microbiological quality.

1. The raw water quality at the E.L. Smith intake as measured by the fecal coliform and the total coliform tests is good. As with all surface water supply, increasing concern with the presence of pathogens which do not correlate well with indicator organisms requires that the best possible process control and performance be maintained at the plant. Sufficient redundancy must be maintained to insure safe water at all times.
2. The raw water quality at the Rossdale intake is of poor quality. It is possible, with the current left bank intake to have very high concentrations of indicator microorganisms in the raw water before treatment. The concerns expressed in item 1 pertain here.
3. The raw water testing should be for the fecal coliforms on a routine, not less than daily, basis. The value of the total coliform test is much lower with regard to identification of fecal contamination.

4. Growth of the City will increase the frequency and amount of contamination reaching the present Rossdale intake. Moving the intake upstream beyond the Groat Road Bridge should result in considerably improved microbiological characteristics of the raw water.
5. The Devon chlorinated wastewater discharge does not appear to reach the E.L. Smith water intake on a regular basis. A variety of possibilities may contribute to the periodic increases in microorganism values. One may be variations in the mixing of the Devon effluent in the North Saskatchewan River.
6. The microbiological quality of the North Saskatchewan River upstream of Edmonton as indicated by the total and fecal coliform numbers does not appear to have changed a great deal since 1973.
7. Recommendations for additional investigations are separated in to two groups, those which are of immediate interest to the City of Edmonton, and those of more general interest for future water quality management.

7.1 Immediate interest.

7.1.1 The storm water discharges need to be defined under different conditions. These should include spring melt; spring, summer, and fall storms; and winter melts.

7.1.2 A detailed description with field verification of transverse mixing conditions through the City is required.

7.1.3 The longitudinal mixing characteristics of an instantaneous discharge needs to be investigated to aid with contingency planning.

7.2 General interest.

7.2.1 Detailed investigation of the microbiological characteristics of spring melt; spring, summer and fall storms; and winter melts.

7.2.2 The relationship between indicator microorganisms and pathogenic microorganisms of various types in various types of storm water discharges is required.

7.2.3 Investigation of means of improving the microbiological quality of storm sewer discharges.

References

1. Northwest Hydraulic Consultants Ltd. and Alberta Environment. 1978. Edmonton Regional Utilities Study, Vol. III, Surface Water. Alberta Environment, Pollution Control, Edmonton, 99p.

2. Anon. 1981. Outfalls. Water and Sanitation, City of Edmonton, 28p. (updated by staff)

3. Ahmad, M. 1985. Status Report on Non-Plant Sanitary Sewer Discharges to the North Saskatchewan River. Water and Sanitation, City of Edmonton, 29p.

4. Ward, J.G. 1986. Prevention of Run-Off From Combined Sewer System From Going Into The North Saskatchewan River Water and Sanitation, City of Edmonton, 6p.

5. Environment Canada. NAQUADAT, Inland Waters Directorate, Water Quality Branch, Burlington.

6. op. cit. 1980. Detailed Surface Water Quality Data, Alberta, 1974-76. Inland Waters Directorate, Calgary. 407p.

7. op. cit. 1982. Detailed Surface Water Quality Data, Alberta, 1977-79. Inland Waters Directorate, Calgary. 388p.

8. op. cit. 1984. Detailed Surface Water Quality Data, Alberta, 1980-81. Inland Waters Directorate, Calgary. 164p.

9. op. cit. 1985. Detailed Surface Water Quality Data, Alberta, Manitoba, Northwest Territories, and Saskatchewan, 1982. Inland Waters Directorate, Regina. 275p.

10. Bell, J.B. 1975. The Bacteriological Content of the North Saskatchewan River in the Vicinity of Edmonton, Alberta. Environmental Protection Service Rep. No. EPS-5-NW-75-2, Edmonton, 29p.

11. Zaal, J.F., J.B. Bell, W. Macrae, and A. Boyko. 1976. Water Pollution in a Prairie River. The North Saskatchewan River in the Vicinity of Edmonton, Alberta, 1975-76. Environment Canada, EPS. Unpublished manuscript.

12. Paetkau, P. 1970. Pollution Survey: Oldman River, Bow River, Red Deer River, North Saskatchewan River, 1969-1970. Pollution Report No. 1, Province of Alberta, Fish and Wildlife Division, Department of Lands and Forests, Edmonton, 31p.

13. Paetkau, P. 1971. Summary Report Biological Pollution Surveys: Oldman River, Bow River, Red Deer River, and North Saskatchewan River, 1970-1971. Province of Alberta, Department of Lands and Forests, Fish and Wildlife Division, Edmonton, 16p.

14. Kupchanko, E.E. 1969. Summary Report North Saskatchewan River Pollution Survey 1968-1969. Province of Alberta, Department of Health, Environmental Health Services Division, Edmonton, 63p.

15. Kupchanko, E.E. 1970. Summary Report North Saskatchewan River Pollution Survey 1969-1970. Province of Alberta, Department of Health, Environmental Health Services Division, Edmonton, 62p.

16. Masuda, A. 1971. Pollution Survey Summary North Saskatchewan River 1970-1971. Province of Alberta, Department of the Environment, Edmonton, 119p.

17. Masuda, A. 1972. Water Quality North Saskatchewan River 1971-1972. Province of Alberta, Department of the Environment, Edmonton, 58p.

18. Stanley Associates Engineering Ltd. 1975. North Saskatchewan River Water Quality Study. Data Appendix. Alberta Environment, Edmonton, 21p.

19. Thomas, J.F.J. 1956. Industrial Water Resources of Canada, Saskatchewan River Drainage Basin, 1951-52, Water Survey Rep. No. 7, Department of Mines and Technical Surveys, Ottawa. 154p.

20. Reynoldson, T.B. 1983. North Saskatchewan River Water Quality 1970-1981. Alberta Environment, Edmonton, 34p.

21. Anon. 1983. Surface Water Quality in Major Alberta Rivers, Fall-Winter 1982/83. Alberta Environment, Pollution Control Division, Edmonton, 113p.

22. Gore & Storrie Ltd. and Hydroqual Consultants Inc. 1986. Water Quality Study, North Saskatchewan River Basin, Phase I. Planning Division, Alberta Environment, Edmonton, 192p.

23. City of Edmonton, Water and Sanitation. 1981. Water Treatment Plants, 1980 Annual Report, Edmonton. 73p.

24. _op. cit_. 1982. Water Treatment Plants, 1981 Annual Report, Edmonton. 102p.

25. _op. cit_. 1983. Water Treatment Plants, 1982 Annual Report, Edmonton.

26. _op. cit_. 1984. Water Treatment Plants, 1983 Annual Report, Edmonton, 121p.

27. _op. cit_. 1985. Water Treatment Plants, 1984 Annual Report, Edmonton. 136p.

28. _op. cit_. 1985. Water Treatment Plants, January to November Monthly Reports, Edmonton.

29. AWWA Committee on Bacteriological Sampling Frequency in Distribution Systems. 1985. Committee Report: Current Practice in Bacteriological Sampling. Journ. Am. Water Works, Assoc., 77, 9, 75-81.

30. Edmonton Journal. 1983. Various dates, autumn of 1983.

31. Federal-Provincial Working Group on Drinking Water. 1979. Guidelines for Canadian Drinking Water Quality 1978. Minister of National Health and Welfare, Ottawa, Ontario, 76p.

32. Anon. 1984. Guidelines for Drinking-Water Quality, Volume 1, Recommendations. World Health Organization, Geneva, Switzerland, 130p.

33. Anon. 1977. Alberta Surface Water Quality Criteria. Water Quality Br., Standards and Approvals Division, Alberta Environment, Edmonton, 17p.

34. Anon. 1978. Recommended Standards for Water Supply and Sewage. Standards and Approvals Division, Alberta Environment, Edmonton, 49p.

35. Anon. 1976. National Interim Primary Drinking Water Regulations. U.S. Environmental Protection Agency, Office of Water Supply, Washington, D.C., 159p.

36. Public Health Service. 1969. Manual for Evaluating Public Drinking Water Supplies. U.S. Department of Health, Education and Welfare, Cincinnati, Ohio, 62p.

37. Public Health Service. 1962. Drinking Water Standards, Revised 1962. U.S. Department of Health, Education and Welfare, Washington, D.C., 61p.

38. Anon. 1968. Water Quality Criteria. Federal Water Pollution Control Administration. Washington, D.C., 234p.

39. Anon. 1973. Water Quality Criteria 1972. National Academy of Sciences, Washington, D.C., 594p.

40. Anon. 1976. Quality Criteria for Water. U.S. Environmental Protection Agency, Washington, D.C., 256p.

41. Lippy, E.C. and S.C. Waltrip. 1984. Waterborne Disease Outbreaks - 1946-1980: A Thirty-five Year Perspective. Journ. Am. Water Works Assoc., 76, 2, 60-67.

42. IHD-WHO Working Group on Quality of Water. 1978. Water Quality Surveys, A Guide for the Collection and Interpretation of Water Quality Data. UNESCO-WHO, Geneva, Switzerland, 350p.

43. Geldreich, E.E. 1966. Sanitary Significance of Fecal Coliforms in the Environment. U.S. Fed. Water Pollution Control Administration, Cincinnati, Ohio, 122p.

44. Geldreich, E.E. 1967. Fecal Coliform Concepts in Stream Pollution. Water and Sewage Works - Reference Number, 114, R98-R110.

45. Geldreich, E.E., L.C. Best, B.A. Kenner, and D.J. Van Donsel. 1968. The Bacteriological Aspects of Stormwater Pollution. Journ. Water Poll. Control Fed., 40, 11, Part 2, R1861-R1872.

46. Geldreich, E.E. and B.A. Kenner. 1969. Concepts of Fecal Streptococci in Stream Pollution. Journ. Water Poll. Control Fed., 41, 8, Part 2, R336-R352.

47. Geldreich, E.E. 1970. Applying Bacteriological Parameters to Recreational Water Quality. Journ. Am. Water Works Assoc., 62, 2, 113-120.

48. Feachem, R.G., D.J. Bradley, H. Garelick, and D.D. Mara. 1983. Sanitation and Disease Health Aspects of Excreta and Wastewater Management. John Wiley & Sons, Toronto, 501p.

49. Oliveri, V.P., K. Kawata, and C.W. Kruse. 1978. Relationship between indicator organisms and selected pathogenic bacteria in urban waterways. Prog. Wat. Tech., 10, 361-379.

50. Whipple, W., N.S. Grigg, T. Grizzard, C.W. Randall, R.P. Shubinski and L.S. Tucker. 1983. Stormwater Management in Urbanizing Areas. Prentice-Hall, Inc., Englewood Cliffs, New Jersey. 234p.

51. WHO Scientific Group. 1979. Human Viruses in Water, Wastewater and Soil. World Health Organization, Geneva, Technical Report Series 639. 50p.

52. Environment Canada. 1979. Water Quality Sourcebook. Inland Waters Directorate, Water Quality Branch, Ottawa. 88p.

53. Logsdon, G.S., J.M. Symons, and R.L. Hoye. 1979. Water Filtration Techniques for Removal of Cysts and Cyst Models. Waterborne Transmission of Giardiasis. United States EPA Report EPA-600/9-79-001. 240-256.

54. Logsdon, G.S., V.C. Thurman, E.S. Frindt, and J.G. Stoecker. 1985. Evaluating Sedimentation and Various Filter Media for Removal of Giardia Cysts. Journ. Am. Water Works Assoc., 77, 2, 61-66.

55. Cabelli, V.J., A.P. Dufour, L.J. McCabe and M.A. Levin. 1982. Swimming-Associated Gastroenteritis and Water Quality. Am. Journ. of Epidemiology, 115, 4, 606-616.

56. Hutcheon, B.C. and D.W. Smith. 1981. Raw Water Studies Determine Siting of New Intake. Journ. Am. Water Works Assoc., 73, 16-23.

57. Beltaos, S. 1979. Transverse mixing in natural channels. Canadian Journ. for Civil Engineering, 6, 575-591.

58. Putz, G. 1984. TRSMIX (Transverse mixing computer program) Users Manual. Environmental Engineering Technical Report 84-2, Department of Civil Engineering, University of Alberta, 64p.

59. Beltaos, S. and M.D. Anderson. 1979. Mixing characteristics of the North Saskatchewan River below Edmonton: Part 1. Report No. SWE 79/01, Civil Engineering Department, Alberta Research Council, 50p.

60. Ahmad, M. 1985. Wastewater Management Alternatives for Improving the North Saskatchewan River Water Quality. Water and Sanitation Department, City of Edmonton, Alberta, 91p.

61. Putz, G., R. Gerard and D.W. Smith. 1983. Effluent dilution and decay, Slave River downstream of Fort Smith, N.W.T. Environmental Engineering Technical Report No. 83-1, Department of Civil Engineering, University of Alberta, 150p.

8. Appendix A

Effluent Mixing Analyses

Introduction

It may be possible to achieve a marked improvement in Edmonton's water quality by moving the water intake away from the bank and hence away from the plumes shed by the various stormwater and sanitary sewer outfalls that discharge into the North Saskatchewan River upstream of the intake. To investigate this possibility analytical simulations of the effluent plume characteristics were undertaken.

There are some 85 sewer outfalls within the City limits upstream of the present water intake. For the present study separate simulations for each outfall would be an unnecessarily onerous task. Instead advantage was taken of the linear nature of the mixing process, and the associated validity of superposition, to construct a series of influence lines from solutions for the concentration distributions in effluent plumes originating at the four selected reference locations along the reach of interest shown in Figure A1. These influence lines allow direct determination of effluent concentration at the intake section caused by an outfall located anywhere along the reach of interest. The concentration due to each outfall of interest can then be determined from these influence lines and simply added together, or superimposed, to get the cumulative effect of the outfalls on the effluent concentration at the water intake section.

Mixing Analysis

The concentration distributions within the plumes originating from the reference locations were determined by solving the differential equation describing the development of a plume from a steady (ie. not changing with time) effluent discharge viz.

$$\frac{\partial C'}{\partial x} = \frac{1}{Q^2} + \frac{\partial}{\partial 7} (uh^2 E_z \frac{\partial C'}{\partial 7}) \qquad (A1)$$

where C' is the dimensionless, or relative, concentration given by

$$C' = \frac{C}{C_\infty}, \qquad (A2)$$

C_∞ is the 'fully mixed' concentration given by

$$C_\infty = \frac{\overset{o}{m}}{Q}, \qquad (A3)$$

$\overset{o}{m}$ is the rate of discharge of pollutant from the outfall, x is distance downstream of the outfall location, n is the dimensionless discharge q/Q where

$$q = \int_o^z uh \, dz \qquad (A4)$$

the discharge passing between the left bank and the point of interest across the stream a distance z from the left bank, u is the depth-averaged velocity and h the depth at the point z, Q is the total river discharge and E_z is the estimated mixing coefficient for the reach of interest. The cumulative discharge q is simply another way of representing transverse position across the river at any given section. In a natural stream it is a much more meaningful and convenient measure than the transverse distance z.

The derivation of Equation A1 is described in detail by Putz (58). It assumes the effluent is fully mixed over the depth of flow, a fair assumption for sections more than about 100 depths downstream of the outfall. The numerical algorithm used to solve this equation is contained in the program TRSMIX, and a supplementary program TRSMIX, DV, programs developed in connection with mixing studies on the Slave River described by Putz, Gerard and Smith (61). Details of the programs are given by Putz (58).

In addition to a suitable numerical algorithm, solution of this equation requires definition of the hydraulic geometry of the reach at the discharge of interest, and an estimate of the mixing coefficient. Cross sections of the river taken in 1972 were available from River Engineering Branch, Alberta Environment. Of the many cross sections available nine were selected as being sufficiently representative of the reach for the purposes of this study. The section locations are shown in Figure 4.1. As this is a gravel-bed reach with a controlled discharge (by the two dams upstream) little change in channel geometry is likely to have taken place since 1972. This contention is supported by the fact that the relation between discharge in the river and water level at the Water Survey of Canada (WSC) gauge at the Low Level Bridge has not changed significantly.

The waterway geometry, and hence the effluent plume characteristics, change with river discharge. As the widest plumes occur at low discharges, a suitably low discharge of 150 m^3/s was chosen as the 'reference' discharge in this preliminary study. This discharge is a little larger than the highest annual minimum flow that has occurred since the Big Horn Dam was commissioned. No allowance was made for the gradually increasing river discharge that would occur due to the contributions from the outfalls through the City. For the reference situation the river was assumed to be free of ice as again this gives a wider plume.

Because of the low discharge and the irregular bed profile, the water level at each section could not be even roughly estimated by assuming uniform flow. Instead the water levels for the reference situation were determined by carrying out a 'backwater' calculation for the selected discharge using the program HEC2, a standard non-proprietary program issued by the U.S. Army Corps of Engineers. First the program was calibrated using the known water levels at each section determined during the cross section surveys. The discharge at this time was 759 m^3/s. Beginning with the water level given for this discharge at the WSC gauge, the roughness coefficient, Manning 'n', of the reach was adjusted until the HEC2 program adequately simulated the measured water levels at the other sections. The optimum value of n was found to be 0.028, a reasonable value for a reach of this nature. However Kellerhals, Neill and Bray (1972) indicate that the value of n in this reach decreases substantially with discharge. This is unusual, but if it is so the value of n to use at the reference discharge of the present study should be somewhat less than that found above. A value of 0.024 was used. This corresponds to a hydraulic roughness of the bed of 47 mm, or about the D_{84} of the bed material in this reach, which defines a reasonable minimum for the hydraulic roughness of a gravel-bed reach. The required distribution of q across each section is calculated by TRSMIX.DV. It is assumed the Manning equation applies to the whole section and to each vertical in that section.

Selection of an appropriate mixing coefficient, E_z, is largely subjective at present. Field measurements of mixing in the North Saskatchewan River just downstream of Edmonton, described by Beltaos and Anderson (59), gave an average dimensionless mixing coefficient ($E_z/h\sqrt{ghS}$) of about 0.2, where h is the mean channel depth, g is the acceleration due to gravity and S is the water surface slope. However much of the test reach in this case was straight. The trend in the plume development in the portion of the reach that has pronounced curvature suggests a higher value of the coefficient would be more appropriate for a reach such as that upstream of the City intake. Furthermore, the measured mixing coefficient reported by Beltaos (1979) for the Athabasca River downstream of Athabasca, a reach not dissimilar to that of interest herein, was about 0.4. Hence this latter value was accepted as appropriate for the North Saskatchewan River upstream of the City intake.

Use of the program TRSMIX requires specification of the parameters listed in Table A1.

Table A1: Parameters specified for the program TRSMIX

Parameter code	Parameter Description	Values used for reference situation
NXS	Number of cross sections to define reach	9
QF	Total stream discharge	150 m^3/s
USTAR	Values of 'shear velocity' ($\sqrt{gRS}$) for each sub-reach, where R is the hydraulic radius.	Calculated from the hydraulic geometry of each sub-reach.
BETA	Dimensionless mixing coefficient	0.4 for each sub-reach.
RLEN	Mixing length scale (approx. the mean channel depth)	1 m for each sub-reach.
RK	Decay coefficient	Zero
DX	Grid interval along the stream	200 m
DQ	Grid interval across the stream, in dimensionless discharge coordinates	0.02
DZ	Diffusion factor, uh^2E_z, in the vicinity of the outfall	0.011 m^2/s^2
POINT	Transverse location of the effluent source expressed in discharge coordinates	0.02 (left bank) 0.98 (right bank)

Selection of the reference situation was such as to give a reasonable upper bound on the plume width at the water intake, and hence the strongest constraint on the choices for an intake location that would be free of the influence of the effluent plumes shed by the outfalls upstream. To give some indication of the sensitivity of the plume characteristics to the choices made, further solutions of Equation A1 were carried out for an outfall at the same location as the Millwoods outfall but with a higher discharge (300 m^3/s), a lower mixing coefficient (0.2), and for an ice covered river. The effect of the ice cover was simulated by simply increasing the waterway depth by 0.23 m, the increase determined as that being required for an ice cover with an hydraulic roughness of 10 mm on a wide river. In addition, the effect of assuming the outfall acted as a line source that extended some 40 m from the bank, rather than the point source 20 m from the bank assumed for the reference situation, was investigated.

Results and Discussion

The detailed calculations and results include:

1. Calculation of the water levels at the WSC gauge section corresponding to the discharges 150 and 300 m^3/s;
2. Input and output for the HEC2 runs for the above discharges;
3. Calculation of the water level adjustment to allow for ice cover effect;
4. Input and output for the TRSMIX.DV. runs for the selected parameter combinations and the 9 selected cross sections;
5. Input and output for the various TRSMIX runs.

The generalized results and the implications of the various simulations are given and discussed in the main body of the report. However there are several limitations to the analysis that should be kept in mind. An important one is that the pollutant discharge from the outfall is considered steady, whereas in reality it varies over orders of magnitude throughout the day. Hence, although these fluctuations will be smoothed somewhat by dispersion during the travel time to the intake location, the concentrations given by the simulations must be considered as mean values taken over one or two days. Higher concentrations can exist over shorter periods. The steady state solution is also of limited utility for assessing the impact of a short-term spill of contaminant on the water quality at the City intake but, as the plume configurations would be similar, it does give a reasonable indication of the portion of the river that would be effected.

Another simplifying assumption was that discharges from the various outfalls were small relative to the river discharge. While this is reasonably true for each individual outfall, it is not necessarily so for the sum of the discharges from all the outfalls. For example, in the scenario described in Section 4.3, the cumulative outfall discharge is 75 m^3/s, compared to only 150 m^3/s in the river. There are two points to be made about this. First, the important input is the pollutant discharge (eg. fecal coliforms/s) rather than effluent discharge. Second, for the present purposes, it is conservative to neglect the effect of the effluent discharge because, as shown in Figure 4.7, discharge results in a narrower plume.

It is emphasized that the above analysis of the plume configurations is a preliminary one, albeit somewhat better defined than usual for such a reconnaissance study because of the availability of river cross sections and the results of field measurements of the mixing characteristics of the river in a nearby reach. It should also be noted that the analysis was carried out as a component of the microorganism study. If instead the concern was with the impact of a sudden spill of toxic materials into the sewer system, a somewhat different analysis would be required. For example, such a spill could occur in late winter when the river discharge was considerably lower than assumed in the above study. This would result in the contaminant moving along a broader plume located further out in the stream.

Hence recommendation for refinement of the above analysis depend largely on the decision about what to do with the intake and the reason for that decision. If it is to move the intake to avoid exposure to a spill of toxic material the emphasis should be placed on refining the definition of the hydraulic geometry and mixing characteristics of the stream at low discharges; if it is to improve the day-to-day quality of the raw water with the intake at its present location, the emphasis should be on better definition of pollutant-discharge from the outfalls upstream. But, whatever the decision, further refinement of the analysis is required if mixing of pollutants in the river has a significant impact on raw water quality at the future intake site.

APPENDIX B

INTAKE FECAL COLIFORM PREDICTION SCENARIO DEVELOPMENT

The general procedure for development of the scenario are outlined below:

1. Establish the flow of interest and develop the general dimensionless concentration curves for various upstream discharge locations. (see Sections 4.1 and 4.2)

2. Identify and, as necessary, group the outfalls by location and bank.

3. Locate center of discharge volume based on expected flow condition (steady state assumed).

4. Develop expected concentration for each outfall (steady state assumed).

5. Calculate total number or mass of contaminant discharged per second.

6. Calculate completely mixed no loss concentration in river (C_∞).

7. From figures find C/C_∞ values for each outfall or group of outfalls at desired distances across the river.

8. Sum values and plot.

Calculation of Concentration (FC/dL) at Intake.

The first step was to combine the 84 outfalls into 8 groups. Four groups were on the right side (R-I to R-IV) and four on the left side (L-I to L-IV).

Each outfall was classified as to type. The classifications were:

ST - Storm water only
ST + WW - Storm water and waste water
C - Combined sewage
ST + C - Storm water and combined sewage

The diameter of the outfall and its discharge was recorded. Discharge values were available for the "ST + WW" and the "C" outfalls from "Status Report on Non-Plant Sanitary Sewer Discharges by the North Saskatchewan River" (3). These values were the total flow during a 5 year storm. The discharge values for "ST" outfalls were estimated. This was done by comparing their diameter to similar diameter outfalls with known discharge. The approximate size of the area served by the outfall was also used in determination of the discharge.

Estimation of the Scenario Flow

25% of the 5 year storm flow was used for the scenario flow.

Calculation of FC/dL

Values of FC/dL were calculated for each outfall.

Concentrations:

Storm water (ST) = 5×10^4 FC/dL

Waste water (WW) = 2×10^6 FC/dL

Assume:

Storm water outfalls (ST) = 5×10^4 FC/dL

Combination outfalls (D) = $\frac{ST + WW}{2}$

$$\frac{5 \times 10^4 + 2 \times 10^6}{2}$$

So that
Combination concentration = 1.025×10^6 FC/dL

For outfalls. Storm water + waste water (ST + WW) the following formula was used:

$$\text{Concentration for (ST + WW)} = \frac{(Qst \times 5\times10^4 \text{ FC/dL}) + (Qww \times 2\times10^6)}{Qt}$$

Qww was obtained from "Status Report on Non-Plant Sanitary Sewer Discharges to the North Saskatchewan River" (3).

For example,

Calculation for ST + WW

#101 Qt = $0.67\ m^3/s$ Qww = $0.13\ m^3/s$

Qst = Qt = Qww = $0.54\ m^3/s$

$$\text{Conc.} = \frac{(0.54 \times 5\times10^4) + (0.13 \times 2\times10^6)}{0.67} = 4.28 \times 10^5$$

Report on

Edmonton, Alberta Water Supply System

by

Vernon L. Snoeyink, Ph.D.

May 1, 1986

Sources of Information

The drinking water treatment plants for the City of Edmonton are the E. L. Smith and the Rossdale plants. I obtained information about these plants:

1. During my visit on January 13-14, 1986, from City personnel and personal observation,
2. From numerous discussions with Dr. Steve Hrudey,
3. From the article by Walker et al. in Jour. Amer. Water Works Assoc. 78, 84 (1986),
4. From the 1984 "Summary of Chemical and Physical Water Quality Parameters at Treatment Plant Sites" for Edmonton,
5. From the Water Treatment Plants Monthly Report for March, April and May, 1985, for Edmonton.

The information on the Hays Mine Plant in Pittsburgh was obtained during a site visit on January 23, 1986, from operating logs, and from the report "Water Treatment Process Modification for Trihalomethane Control and Organic Substances in the Ohio River," by ORSANCO, USEPA Report 600/2-80-028, March 1980. The information on the St. Louis County Treatment Plants was obtained through personal contact with plant personnel.

E. L. Smith Plant

Source Water Quality

The North Saskatchewan River is the source of water supply. This river is fed by other rivers which flow through muskeg and bushland areas, and during periods of snowmelt and heavy rains, organics causing color, taste and odor are leached out of these areas and make the water difficult to treat. For example, average raw water turbidity and color values were 10 to 26 NTU and 9 to 23 CU, but maximum values were 95 to 300 NTU and 40 to 460 CU, respectively (see Walker et al., 1986).

The finished water odor complaints are not segregated based on whether the water comes from E. L. Smith or Rossdale, but the opinion of plant personnel is that the worst problems for E. L. Smith occur during the March-April snowmelt period. An off-line reservoir that would permit by-pass of this water could be effective, and its cost should be analyzed. The taste and odor frequency tabulation of Walker et al. show frequent odors in other months of 1982-1984, but these may be caused by Rossdale water. The Walker et al. article refers to high color and phenolics-laden water which results from releases from upstream impoundments, but I have not been able to obtain the data which show this.

The general opinion of plant personnel is that there is little, if any, contamination from other than natural sources because the region between Edmonton and the mountains is sparsely populated. This opinion should be verified by analysis; this can best be done in conjunction with a determination of the compounds that form during treatment as recommended below.

Treatment Procedures and Process Control

The E. L. Smith Plant is modern and appears to be well run. The treatment sequence includes addition of ammonia and ClO_2 to raw water, clarification with alum in upflow clarifiers, lime softening in upflow clarifiers, secondary disinfection with chloramines, and filtration through anthracite sand filters. Powdered activated carbon (PAC) and activated silica may also be used. Chlorine dioxide is dosed to maintain a concentration of 0.1 mg/L leaving the plant (see Walker et al.) Chlorine to form monochloramine residual is added just before the filters.

Sources of Odorous Compounds

An important topic on which more information is needed is the source of the odor problems in the finished water. The nature of the odor causing compounds is very important relative to the procedures that can be used to successfully control the problem. For example, earthy-musty odors are often caused by geosmin or methylisoborneol, low molecular weight aliphatic compounds, which are difficult to oxidize but relatively easy to adsorb on carbon. I expect some of these compounds to be present, but we do not have data showing their presence. Odorous compounds may also form during treatment. American Water Works Service Company has had two experiences, at Lexington, Kentucky, and Charleston, West Virginia, where too much ClO_2 pre-oxidant formed "kerosene" type odors. Reduction of ClO_2 dose so that 0.1 to 0.2 mg/L was in the water going onto the filters apparently eliminated the problem. Professor Hoehn of Virginia Polytechnic Institute has observed the formation of "hydrocarbon" or "lighter fluid " type odors from ClO_2 addition at Stafford County, Virginia. Professor Sontheimer of the University of Karlsruhe, FRG, has also observed ClO_2-caused odors in West Germany when ClO_2 was used as a pre-oxidant. The solution in the latter case was to eliminate application of ClO_2 before the filters. Sufficient natural organic matter removal by coagulation, sedimentation, filtration was achieved so that the problem did not exist when ClO_2 was added after the filter. I have not been able to find out much about the ClO_2 reaction which causes these odors, but it is possible that the ClO_2 reacts with humic substances or other natural organics. If humics are the cause of part of the problem, the best approach could be to eliminate as much of the natural organic matter as possible by coagulation-sedimentation-filtration before ClO_2 is added; the ClO_2 could then be added after filtration. The current approach of increasing the ClO_2 preoxidant dose during times of poor water quality could be aggravating the odor problem. PAC has not proven effective for adsorption of humic substances so PAC probably will not prevent odor formation by this reaction, and complete destruction of the humics by chemical oxidation would require too much oxidant to be effective. If chlorine dioxide is not added until after filtration, ammonia addition should be delayed until the time of chlorine addition. Otherwise, nitrification will probably occur in the upflow clarifiers.

There is a strong possibility that odor is forming in the distribution system. This is not well documented because the necessary odor measurements have not been made, but this is an important area of study. Is odor formed in the distribution system? Does monochloramine react with the aqueous organics to produce odor? Is there any free chlorine present in the distribution system, and if so, is it producing the odor? Does the combination of monochloramine and ClO_2 produce odor? Does odor formation take place only in the distribution system, or can it form in reactors in the laboratory? If it forms only in the distribution system, perhaps organism growth in the distribution system is playing a role. These questions must be answered before treatment changes can be suggested.

I recommend a testing program to determine the cause of the problem and the best solution. The odors entering the plant should be characterized according to type and intensity. Extraction and GC/MS analysis of the water entering the plant is necessary to establish a baseline of what compounds are present in the raw water, and whether the source water is free of other than natural organics. The relationship between TON and PAC dose should be established by jar tests. Bench scale tests should also be carried out to determine whether odors are formed by ClO_2 reactions with natural organics, and if so, the rate of this reaction when ClO_2 is added as a pre-oxidant and when it is added only after filtration. PAC does not adsorb humic materials very well because of their large size and slow rate of diffusion, so if they are a cause of the problem, it will be necessary to rely on coagulation or possibly another process to remove them. Optimum conditions for removal of natural organics by coagulation should also be established so that coagulation practice can be modified if necessary. Addition of ClO_2 after filtration would require that there be no problems from the combination of ClO_2 and monochloramine, and that there is sufficient contact time for ClO_2 to act after filtration, before distribution. The conditions causing odor formation, if any, in the distribution system should be established.

PAC

PAC is used extensively at E. L. Smith to control odor. It has proven to be effective in drinking water treatment for removing low molecular weight, odorous organic compounds such as chlorophenols, methylisoborneol, geosmin and other substances, but there are many different grades of it, and there are different ways of applying it. It is not likely to be effective in removing large molecular weight compounds, however, and if odors are produced after the PAC has settled out, from reaction between humics and ClO_2, for example, the PAC will not be able to remove them. It is important to determine which compounds can be removed by PAC at E. L. Smith, and then to do jar tests to determine the most cost-effective PAC. These studies may show that more than 20 mg/L of PAC is needed at times, and thus that it is necessary to increase PAC feed capacity.

The method of applying PAC can also influence its effectiveness. For example, addition of PAC before coagulant is added allows a faster rate of adsorption than if PAC is added at the same time as or after the coagulant. The PAC apparently is incorporated into the floc particles, thus slowing adsorption. For this reason, the potential advantage of adding PAC at the intake, rather than in the rapid mix, should be investigated. Another possibility to be considered is to convert the first clarifier to a PAC slurry contactor during the time of snowmelt problems. No coagulant, or possibly only polyelectrolyte, would be added, and the PAC would be removed by coagulation in the second basin. Softening could be eliminated during this time because a 30 mg/L hardness change is not a significant change to most users. Alternatively, larger doses of PAC to the first basin with modified coagulation may be effective. Because PAC is a good reducing agent, it chemically reduces oxidants such as chlorine and ClO_2, thereby increasing oxidant demand. I expect the increase in oxidant demand to be proportional to the PAC dose when the two substances are allowed to come into contact. In floc blanket reactors, such as are used at E. L. Smith, PAC accumulates in floc blankets, and is retained in the system much longer than it would be in conventional, horizontal flow sedimentation basins such as are in use at Rossdale. The increased time of contact should yield more efficient use of PAC, but the PAC must be incorporated in floc in order to be retained in floc blankets. The necessary research to determine the best way to maximize the effectiveness of PAC in this process has not been done. It is being studied at Newport News, Virginia, by Professor R. Hoehn and Carl Hamann of CH_2M-Hill, and in my laboratory, but no definite statements can be made about the amount of increase in effectiveness now. I encourage E. L. Smith Plant personnel to monitor the concentration of PAC in the floc blanket at different PAC doses, and to make careful observations of its effectiveness under different flocculation conditions.

Because the worst odor problem seems to come at predictable times in the February-April time period, feeding PAC in advance of this period at a dose of a few mg/L should allow PAC to build up in the floc blanket and, thus, to be available when needed. The dosage could then be increased when the poor water quality arrives. This approach should work well for the odorous compounds in the raw water. The study of ways to improve PAC effectiveness should include an examination of the optimum PAC dose--much larger doses than the maximum now possible may be necessary.

Monitoring

Raw water is monitored for turbidity color and UV absorbance. Because conditions of intense odor coincide with high values of these parameters, monitoring them and instituting procedures to control odor when values are high makes good sense.

I am not convinced that the GC monitoring of treated water is providing useful information for process control. Is it measuring compounds that were in the raw water that are odorous and that can be removed by PAC? Or is it measuring compounds that are formed from ClO_2? If the compounds were in the raw water, a reasonable response to high values is to increase PAC dose, but this may not be a reasonable response if the compounds are formed during treatment. The correlation between the TON of raw water odors and GC response needs to be carefully established. Without this quantitative relationship, there is not an adequate means of determining how much carbon to add, or of assessing the effectiveness of a given dose and method of application. It may be possible to develop methods to quantify the concentration of odorous compounds present using the GC, but the current procedure of estimating the concentration of odors based upon a head-space or purge-and-trap chromatogram is not good.

Residual Aluminum

During winter operation, lime and alum are added together for clarification and softening. Use of alum in high pH waters can cause a high aluminum residual concentration in treated water. High aluminum residual can cause turbidity formation and aluminum oxide scale formation in the distribution system, and is undesirable for kidney dialysis. The

European Economic Community has set an MCL of 0.2 mg/L and a guideline value of 0.05 mg/L. AWWA is considering the same values as an operating level and goal, respectively.

Aluminum has minimum solubility in the pH range 5 to 6.5. At higher pH, solubility decreases as pH increases because of the formation of the soluble anion, $Al(OH)_4^-$. The amount of residual aluminum at any pH is a function of the age of the $Al(OH)_3$ precipitate and the process efficiency for removing supersaturated aluminum. In cold weather, process efficiency is often lower because of slow precipitation kinetics, and the poor settleability and filterability of the fine $Al(OH)_3$ particles that are produced. (Further details of the chemistry of aluminum are given in the text Water Chemistry, Snoeyink and Jenkins (John Wiley and Sons, 1980).)

Control of residual aluminum should begin with an analysis of treated water for aluminum throughout the year to determine whether there is a problem. If so, and if modification of the alum coagulation-lime softening process to reduce excessive residual is not possible, it may be necessary to change from alum to a ferric salt coagulant. Ferric salts are much less soluble than alum and are not subject to the same problems at high pH.

Rossdale Plant

The Rossdale Plant is an old plant in the center of the City which has been renovated several times and is now being renovated once again. The modifications include the addition of a rapid mix to Plants I and II for improved coagulation, and filtered water reservoirs to improve disinfection. The addition of the reservoirs is especially important because it provides for more flexibility in operating the plant.

Source Water Quality

The North Saskatchewan River is the water supply source for Rossdale as well as E. L. Smith. Unfortunately, the Rossdale intake is located within the City, below several storm water outfalls, so there are water quality problems in addition to those at E. L. Smith. There are possible sources of organics, such as the storm drains, the snow that is taken from streets and deposited near the river to melt, and other sources related to development along the river.

The chief consumer complaint about the water quality is the odor at different times of the year. Given the unpredictable quality of the source water, the presence of odor should be considered only as one indicator of finished water quality. Treatment should be employed that will remove odor and other potentially harmful compounds, but simply removing, or preventing the formation of odor will not insure a good quality water with respect to other organics.

The alternative of changing the intake location to the E. L. Smith Plant location should be closely investigated. Delivery of better water to the Rossdale Plant would eliminate many concerns about the unknown effects of contaminants that escape treatment. PAC could be added so that adsorption could take place during transport from E. L. Smith to Rossdale if studies showed that PAC added in this manner was effective. Of course, the quality and treatability of the supply at the Smith Plant, which must yet be determined in some respects, and the ability to control upstream industrial and municipal development in the future will be important considerations that will determine whether this is an acceptable alternative.

A portion of the water taken into the Rossdale Plant is heated by passing it through a natural gas power plant. The water temperature is raised about 5°C in winter in order to reduce the frequency of water main breaks. There apparently is a risk of hydrocarbon contamination as the water is heated, however, and it is very costly for the City to pay for the energy required to heat the water. Water main breaks that have been experienced in the past apparently occur where the pipes are "paper-thin" from external corrosion. The breaks are a serious problem, but very large expenditures of money are required each year to heat the water. I believe a detailed investigation is warranted to determine 1) frequency and location of breaks, the condition of the pipe, and type of failure, 2) the cause and remedy for the external corrosion problem, and 3) the cost of correcting the corrosion problem and replacing deteriorated pipes versus the cost of continuing current practice. The practice of heating the water before treatment and distribution may be reducing the frequency of breakage, but may not be cost-effective.

Treatment Procedures and Process Control

Existing Processes and Problems

Treatment at Rossdale consists of adding alum, PAC, ammonia and chlorine dioxide to water as it enters the plant, flocculation, sedimentation of alum solids, addition of lime, flocculation, sedimentation of hardness solids, addition of fluoride and chlorine, and filtration. Monochloramine forms by reaction of chlorine with ammonia to provide a residual for the distribution system. Water will then flow to a clear well (under construction) which will have a contact time in excess of 4.5 hours.

The practice of adding chlorine dioxide, PAC and ammonia at the entrance to the plant along with alum needs to be carefully examined. PAC may be destroying some of the chlorine dioxide, thereby increasing the chlorine dioxide demand. Further, it may be possible to use PAC more effectively if it can be added earlier, at the intake for example, so that it does not immediately become incorporated into alum floc. Addition of chlorine dioxide to the raw water needs to be carefully examined because it may be forming odor, as discussed above. Delay of chlorine dioxide addition until after rapid sand filtration would be desirable if it can be shown that there is sufficient contact time for disinfection in the new clear well (a tracer study to establish residence time would have to be carried out) and if it were shown that chlorine dioxide was reacting with organics in the raw water to produce a hydrocarbon odor. For effective use of both PAC and chlorine dioxide, it is essential that the sources of odors be identified, and the adsorbability of those that can be removed with PAC be established.

Residual aluminum should be monitored in Rossdale effluent as well as at E. L. Smith. Particles of alum floc may remain after sedimentation, and these may be solublized when the pH is raised through lime addition for softening.

There appeared to be substantial solids carry over from the softening process to the filters at the time of my visit, and plant personnel indicated that this is a common occurrence. This carry over is probably caused by inefficient $CaCO_3$ precipitation. Its rate of precipitation depends upon a number of factors including temperature and the amount of solids in suspension for precipitation to take place on. If the temperature is low, especially in combination with a low concentration of solids, the rate of precipitation is slow. In solids contact clarifiers, the solids concentration can be controlled much more easily than it can in horizontal flow basins such as are used at Rossdale. Sludge recirculation can sometimes improve performance and should be studied to determine cost versus benefit received.

Filter effluent quality at Rossdale is worse during times of poor influent water quality. The data for March 1985, a time of many complaints about odor, show that backwash was necessary because turbidity exceeded 1 NTU in nearly every case, whereas the data for May 1985 show longer filter runs and that the filters were backwashed for reasons other than turbidity breakthrough. Average turbidity for March was about 0.4 NTU compared to 0.15 NTU for May. The turbidity for March is high; this amount of turbidity may result in sediment deposition in the distribution system. The sediment may cause high plate counts of bacteria, occasional turbid water for consumers, and may be a cause of odor formation. More attention should be given to procedures for coagulating, flocculating and settling the water during times of poor water quality. A detailed jar testing program seems necessary to determine what modifications can best be made, especially during times of rapid raw water quality changes, to reduce turbidity going on to the filters.

PAC is used to remove trace organic compounds at Rossdale. It is not fed continuously, but only when raw water quality is poor as indicated by turbidity, UV adsorbance and GC analysis of volatile compounds. PAC has not been completely effective as indicated by the large number of complaints at various times of the year. PAC is well known for its ability to remove many compounds that cause odor, and other substances such as pesticides. Other substances, such as volatile organic chemicals, THM precursors and TOC, and are not well adsorbed by PAC. Thus, successful removal of odor by PAC will not insure the removal of many other trace organic compounds. Studies at Rossdale are needed to show what compounds are in the water that are removed by PAC, what PAC doses are needed, and whether there are ways to make PAC more effective. Increasing the PAC feed capacity may be necessary, and it may be possible to find a better grade of PAC. I anticipate that such studies will show PAC should be fed continuously because undesirable compounds may be in the water at times when there is no odor, or high turbidity, or high UV absorbance.

Possible Treatment Plant Modifications

Long-term continued use of North Saskatchewan River water from the Rossdale intake will require that significant modifications in treatment be made because of the quality of the water. There are many alternative process sequences that should be investigated in detail, but it is probable that some sequence of ozonation, GAC, biological treatment and disinfection would create a high-quality finished water. Installation of these processes at Rossdale would, of course, require a major revamping of the plant, and in all likelihood expansion of treatment capacity at E. L. Smith would be a better choice.

Assuming that a raw water transport line will be constructed to E. L. Smith intake site, process modifications can be made at Rossdale for the water that will come from the existing intake for the near term and from the E. L. Smith site for the long term. Short term modifications probably would consist only of modifications in chemical addition, as described above, and process monitoring to assure maximum efficiency of existing processes is achieved. If and when major construction is needed, an engineering investigation will be required to determine if the Rossdale or the E. L. Smith site should be used.

An interim measure to be considered is the replacement of the rapid filtration media with granular activated carbon (GAC). Evaluation of the relative cost of using PAC versus GAC for odor removal has shown that if the incidence of odor is relatively frequent, use of GAC as a filter media, on a throw-away basis, is more economical than using PAC. There are very many successful applications of GAC as combined filtration media and an adsorbent for odor; turbidity removals for a good design are equal to or better than sand media. Because of the relatively short contact time available, TOC, THM's, and possibly other compounds, which adsorb slowly, are not effectively removed. Specific turbidity removal and adsorption efficiency should be determined by on-site tests, however. My current research has shown that there can be problems with this application. Because GAC is not as dense as anthracite or sand, it is more difficult to backwash. In applications where adequate surface wash or air scour is not available there have been problems with mud balls. Care must be given to select the proper carbon size (effective size, uniformity coefficient) for adsorption, filtration, and backwash. Underdrain design is also important because GAC particles in the distribution system can aggravate bacterial slime problems.

Desorption should not be a problem if the GAC filter is properly monitored. The appearance of a compound in the effluent at a concentration greater than the influent will not occur as long as there is adsorption capacity remaining for that compound. Thus, if it is known that a particular compound must be removed, a carbon filter must be monitored for that compound, and the carbon must be replaced when the concentration of that compound in the effluent the filter is a fraction (0.2-0.3) of the influent concentration. When a compound has been adsorbed and the influent concentration decreases substantially, desorption of that compound may occur over time, but its concentration will always be less than the concentration in the influent at the time of adsorption. Further, if it is known that a concentration above a certain level is harmful, the column can be monitored for that compound and the carbon replaced when the effluent concentration reaches its undesirable limit.

I regard the use of GAC as a filtration media at Rossdale only as an interim measure because the quality of water is poor, and we need to be concerned about effective removal of compounds in addition to those which cause odor. This could best be accomplished in the long term by GAC in the post filter adsorbers.

Alternatives to chlorine dioxide should also be examined if it is shown that chlorine dioxide is producing undesirable compounds. Ozone and permanganate should be investigated.

Monitoring

Taste and Odor

The current approach to monitoring organics at Rossdale is to attempt to measure the compounds causing odor. There seem to be some serious problems related to this measurement as indicated by the monthly report for March 1985. This report shows that musty water is being produced at Rossdale although it does not indicate the intensity of this odor. The reservoir samples for this month show that all taste and odor measurements were inoffensive. Based on this report one would conclude that some musty water was leaving the plant, but it was only slightly musty, and the problem cleared up by the time the water got to the various distribution reservoirs. The large number of consumer complaints does not agree with this conclusion. The procedure that is followed in the plant for measurement of odor is to continuously spray a heated stream of treated water into a large glass container and to smell the gases that are produced. I think this device can be used to give operators a quick indication of whether problems exist with the treated water, but it is unsatisfactory for providing the quantitative information that is needed to adjust PAC dose or otherwise modify treatment procedures.

The second procedure involves measurement of the volatile organics that are in water using the head space and purge and trap techniques with gas chromatography. This procedure involves processing the sample and making a quantitative estimate of the volatile compounds that are present based upon inspection of the chromatogram. Based upon this estimate, 1) no action may be taken, 2) PAC may be added to the water at varying doses, and 3) if the values are high enough, the E. L. Smith Plant may be shut down, and production at Rossdale may be reduced. This procedure is deficient because of the difficulty of making a good quantitative estimate, and because it has not been established that the compounds that are measured are responsible for odor. It is also not known whether the compounds that are measured are those which are originally present in the raw water, or whether they are formed during treatment. It is also not known how the organics measured by this technique relate to odors that might be formed in the distribution system. Thus, this measurement is not very helpful for controlling the odor problem that exists. With the appropriate supplemental studies, however, it may be possible to obtain good process control information from it.

Measures to control taste and odor are taken when a report of an oil spill is received or when oil is observed on clarifiers and flocculators. These procedures are important parts of plant control because certainly if the contaminants are visible, immediate action must be taken.

Chlorine Dioxide

Control of chlorine dioxide application has been poor. Analytical procedures should be used which measure the ClO_2^-, ClO_2, ClO_3^- and residual chlorine concentrations after ClO_2 is added. The same procedures should be used to measure the yield of the ClO_2 generation. The ClO_2^- and ClO_3^- concentrations are especially important because of the health concerns about them.

Required Modifications in Monitoring

Improvements in organic monitoring procedures are needed, but the specific changes should be developed as part of a study to determine the source of the compounds, and the effects of treatment on their concentrations. For example, the study to improve taste and odor control, discussed above, should include the development of sampling and analysis techniques which will yield information on the type of odor and its intensity. The objective should be to develop techniques that allow odor measurements to be related to consumer complaints. The sampling procedures and frequency of measurement will depend upon the source of the odors.

Monitoring and control of potentially harmful nonodorous compounds at Rossdale also is extremely important. Again, it is not possible to specify a monitoring procedure without studies to better define the problem and to determine which measurements will yield the most information. These studies should include specific compound measurement by GC/MS, including both volatile compounds and those which can be extracted from water using various separation techniques. Samples for analysis should be taken at various points in the treatment system, and should show not only what compounds are present, but also should establish a baseline of removal efficiency of the different processes. Because GC/MS can be used to determine only a small fraction of the total number of compounds which are present, additional parameters should also be used, including TOC, TOX, and possibly other parameters such as total organic nitrogen which might be related to formation of odor in the distribution system. The use of mutagenicity tests should also be considered for use, at least during the study to determine the appropriate monitoring procedures and treatment modifications. Mutagenicity tests are difficult to interpret on an absolute basis, but changes in mutagenicity as a result of treatment are important to observe. One objective for treatment at Rossdale might be to produce a water with no more mutagenicity than E. L. Smith Plant water.

Water Treatment at U.S. Utilities

Hays Mine Station, Pittsburgh, PA

The Hays Mine Water Treatment Plant is part of the Western Pennsylvania Water Company, a division of the American Water Works Service Company. It treats typically 30 to 45 MGD (110-170 ml/per day). The source of supply is the Monongahela River in Pittsburgh. The plant is located in an urbanized area, and the intake is downstream of industrial discharges and other sources of pollution, similar to the situation at the Rossdale Plant in Edmonton. Water quality data for the raw and finished waters are given in Table I. By comparison, the water is softer and lower in alkalinity than the Edmonton water, but raw water turbidities appear similar. Typical TON values in the raw water range from 5 to 9, and values as high as 60 have been measured. A primary source of contamination is an upstream discharge from a steel mill which contains both phenol and benzene. Steel mill personnel are responsible for monitoring the discharge for benzene and phenol and for notifying the water plant when high values are expected. Benzene values in the raw water as high as 80 μg/L are experienced.

The treatment processes include lime addition for pH adjustment, coagulation using cationic polymer only, flocculation and sedimentation, and filtration with GAC as the filter media. PAC can be added at the intake and at the treatment plant. The maximum dose at the intake is 25 mg/L. There is also provision for adding potassium permanganate, and they have experimented with chlorine dioxide in the past. Both pre-chlorination and post-chlorination is practiced. As shown in Table I, treated water quality is very good with respect to turbidity and odor, values in excess of 0.2 NTU are very rare, and no observable odor was found in the effluent of the GAC filters throughout the 1985 year.

The treatment philosophy for controlling organics contaminants is that PAC must be used to protect the GAC as much as possible. PAC is added only at times of high phenol or benzene concentrations, but there were only a few weeks throughout 1985 that no PAC was used. Typical doses are 10-15 mg/L, but doses as high as 36 mg/L were used.

The GAC filters have 30 inches of 12 x 40 F-400. Current data show that a three year old filter is still removing benzene peaks, although measurable levels of benzene can be found in the effluent. I suspect that biological oxidation is removing the benzene. Biological oxidation is also possible for odor removal although there is no evidence for this other than the very long life of the carbon. Data collected when the carbon was new showed that it was effective at removing trihalomethanes for only about 50 days.

I do not have data on whether odor is re-formed in the distribution system. I was also not successful in obtaining cost data for individual processes, but it is interesting to note that the water charge is $2.05 per thousand gallons.

Table I. Raw and Finished Water Quality at Hays Mine, Pittsburgh, PA

	Raw	Finished
pH	7.1	7.3
Alkalinity (mg/L as $CaCO_3$)	20	35
Hardness (mg/L as $CaCO_3$)	130	140
Turbidity	(5-20)[1][4-380][2]	(.15-.2)[1][.1-4.5][2]
Odor (TON)	(5-9)[1.2-60]	none observable
Chlorine Residual (mg/L as Cl_2)		1.1 (combined)

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1. Typical values for 1985

2. Minimum-maximum values for 1985

St. Louis County Water Company, St. Louis, MO

The St. Louis County Water Company serves water to the suburbs of St. Louis. Total treatment capacity is 357 MGD, and average production is 142 MGD. There are five treatment plants at two different locations on the Missouri River, and there are five treatment plants at two different locations on the Meramec River. The former processes 83% of the water while the latter treats 17%.

The raw water quality is given in Table II. One important difference from Edmonton water is the very high turbidity of the raw waters. The average turbidity of the Missouri River is 425 NTU, for example, and in 1984 a peak concentration of 2,600 NTU was reached. The 1984 average color of the Missouri was 16 color units, and the peak color was 35. The raw and finished water is routinely analyzed for specific compounds by GC/MS. It is interesting to note that the specific compounds causing taste and odor have not been identified. Several pesticides have been measured, although no special treatment is employed to remove them.

The treatment sequence at the plants typically includes a large pre-sedimentation basin with detention times ranging from 1 to 3.5 hours; a primary coagulation, flocculation, sedimentation stage with sedimentation times ranging from 4 to 10 hours; and a secondary coagulation, flocculation, sedimentation stage with detention times in the sedimentation basin ranging from just under 7 to 17 hours. Secondary sedimentation is followed by filtration through sand or anthracite-sand filters. The total time to treat the water varies from 17 to 31 hours at four of the larger plants. The chemicals that are added include lime in

Table II. Average Raw Water Characteristics, St. Louis County Water Co.

Characteristics	Missouri River	Meramec River
Calcium, mg/L as Ca	59.2	36.6
Magnesium, mg/L as Mg	19.0	20.0
Ammonia, mg/L as NH_3	0.10	0.05
Fluoride, mg/L as F	0.35	0.10
Turbidity, NTU	425	47
Alkalinity, mg/L as $CaCO_3$	160	151
Total Hardness, mg/L as $CaCO_3$	226	174
Non-carbonate Hardness	66	23
Color	16	15
Total Solids, mg/L	797	247
Dissolved Solids, mg/L	397	207
Suspended Solids, mg/L	400	40
pH	8.3	8.1
Coliform per 100 ml	17,000	6,900
Fecal Coliform per 100 ml	1,400	550
Fecal Streptococcus per 100 ml	7,200	2,800
Standard Plate Count per ml	27,000	17,000

advance of the pre-sedimentation basin to raise the pH to the range of 10 to 10.5, lime, ferric coagulant, fluoride and chlorine in advance of the first stage of coagulation, ferric coagulant in advance of the second stage of coagulation and filter aids consisting of polymers and ferric coagulant prior to filtration. Ammonia usually is added before the filters, and chlorine after the filters, and chloramine is used as the residual disinfectant in the distribution system. The very unique feature of treatment is the very long detention times in the basins.

When odors are not a problem, chlorine and ammonia are added near the entrance to the plant so that a chloramine residual is carried through the treatment plant. However, when odor and color are present, free chlorine on the order of 2 to 2.5 mg/L is carried through the plant, and this residual is converted to monochloramine at the time of filtration. Apparently the free chlorine oxidizes the color as well as the odorous compounds. The long detention times in the basins in the treatment plants often result in the growth of algae, so copper sulfate is applied whenever algae growth is apparent. Powdered carbon is also sometimes used to prevent algae growth by preventing light penetration.

The quality of the finished water seems reasonably good. The average finished water turbidity is less than 0.15 NTU. It is interesting to note that St. Louis County has observed that achieving low finished water turbidity also helps eliminate taste and odor. They report that turbidity greater than 0.2 NTU has been associated with the development of taste and odors in the distribution system 24 to 48 hours later.

The primary warning of taste and odor problems is taste and odor testing several times a day at the point of sample collection. Raw water samples are chlorinated and tasted also. Once a day, samples from all plants are delivered to the central laboratory for taste and TON evaluation under controlled conditions. Finished water TON averages 1.2. Finished water from each plant that has been stored for 24 hours is also evaluated by the taste panel.

St. Louis County has observed that the level of the river, the raw water turbidity and the ammonia concentration in the raw water are all commonly related to odor problems.

Free chlorine is used for elimination of the woody-earthy type odors experienced during the summer. A typical chlorine dose for this treatment is 5 mg/L. This high concentration of chlorine at the high pH that exists in the treatment basins probably results in high levels of THMs. PAC dosages of 4 to 12 mg/L are also used. Carbon will be dosed during a taste and odor episode in both the primary and the secondary coagulation zones. It has been observed that additional chlorine is quite often required to maintain chlorine residual during the time of PAC application. Potassium permanganate has been tried but has not been found to be effective.

St. Louis County receives an average of 350 taste and odor complaints in a year. It is their opinion that 350 is a relatively low percentage of their total customers. Further, it is observed that their taste and odor problems usually occur and disappear within 48 hours. The worst taste and odor on the Missouri River occurs during the time of low river flow in January, February and March.

Activated carbon is applied approximately 50 days per year at each of their plants. Its cost is typically 5% of the total chemical costs per year. However, because additional chlorine and ferric sulfate coagulant must be used when carbon is applied, a dose of 12 mg/L of carbon nearly doubles the cost of chemicals. St. Louis County charges $2.22 per thousand gallons for their finished water.

Summary

The principal water quality problem at E. L. Smith is odor which is difficult to control by the treatment procedures that are now used. The same problem exists at Rossdale, but water quality at Rossdale appears substantially worse because of upstream waste discharges. Specific process changes to solve these problems have not been recommended because not enough is known about the nature of the problem. Recommendations are made for studies to better define the problem, and to determine how the problem can be controlled through treatment. These studies should lead to the necessary process changes to improve treated water quality. Organic compounds are controlled at Hays Mine in Pittsburgh, PA, using PAC and GAC combined, while oxidation with chlorine and PAC are used by St. Louis County. Their treatment procedures are of much interest because they may have application at Edmonton. Caution in using information from these plants is warranted, however, because of what appear to be the unique aspects of Edmonton's problem.

Recommendations

Several recommendations have been made in the report which are repeated here for emphasis.

1. The sources of odor at both E. L. Smith and Rossdale need to be determined. The type and intensity of odor in the raw water, that formed during treatment and that formed in the distribution system, if any, needs to be established as a basis for more effective treatment and monitoring to control the problem. GC/MS should be used in this study to show what compounds are formed during treatment and to establish the types and concentrations of compounds that are in the raw waters at both sites. Once the problem has been properly characterized, process modifications to correct the problem should be examined.
2. The current approach to monitoring odor needs to be modified to include intensity of odor, TON, as well as type of odor. Sampling and analysis for odor should be modified to establish procedures that allow correlation of measured odors with consumer complaints. The use of GC in addition to TON measurements can potentially yield much useful information, but correlations are needed between compounds measured by GC and odor type and intensity.
3. Procedures must be established for monitoring nonodorous organics at Rossdale. The source water quality is very poor and more detailed information on specific compound presence and removal by existing processes is necessary. Group parameters such as mutagenicity should also be considered for use. Development of improved

monitoring procedures will not be as necessary if the decision is made to move the intake to a relatively unpolluted source.

4. Process modifications should be examined at both Rossdale and E. L. Smith. Possible changes include changing the point of PAC addition to the intake to improve removal of compounds in the raw water, delaying addition of chlorine dioxide until after the filter, delaying addition of ammonia until the time of chlorine addition, changing the type of PAC, and possibly others. If chlorine dioxide proves incompatible with the compounds in the raw water, a change to ozonation may be appropriate. Further, efforts should be made to improve coagulation and sedimentation in order to reduce the solids load to the filter and to reduce effluent turbidity during times of poor influent water quality. Elimination of softening to give greater treatment flexibility is a possibility. High effluent turbidity can cause degradation of water quality in the distribution system.
5. Aluminum residual should be measured in the treated water, and if excessive, process modification should be undertaken to control it.
6. Careful consideration needs to be given to the source of Rossdale water. One option is to take water from the E. L. Smith site and transport it to Rossdale. Consideration of this option should include quality control during transport; PAC addition during transport is also a possibility.
7. If the decision is made to continue the use of the existing intake at Rossdale, studies are needed to determine which sequence of processes should be used to provide an effective barrier to passage

of organic compounds through the treatment plant. Alternatives such as ozonation and GAC with biological treatment should be carefully considered, and detailed studies are needed to determine appropriate design parameters and cost of alternatives. If the decision is made to move the intake but construction is not possible for some time, it may be appropriate to consider GAC as a replacement for sand and coal in the existing filters.

8. The cost effectiveness of heating water at Rossdale to prevent water main breaks needs to be carefully studied. Reasonable alternatives may be to control external corrosion, and to replace badly corroded pipes before they break.

9. The recommended studies and increased monitoring cannot be done without increasing the laboratory and engineering staff, and increasing the capability of the laboratory. One approach is to employ a process development and problem solving team to carry out the recommended studies and then to determine the appropriate modifications. Such a team should include a chemist and a process engineer, and possibly a microbiologist. The recommended studies are complex, and good quality people will be required to conduct them. I also recommend an advisory committee of appropriate experts be set up to assist in development of a study plan, and to review the results as they are obtained and the recommendations that are made. This approach will insure that the best possible solutions are implemented.

EDMONTON WATER STUDY

MICROBIOLOGICAL ASPECTS

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3.2.1 Evaluate bacterial indicators and pathogens

3.2.1.1 Review in detail the specific test procedures including sample collection, handling preparation and analysis for all bacterial analyses performed by Edmonton Water and Sanitation and by the Provincial Laboratory of Public Health for drinking water surveys conducted on Edmonton water.

Edmonton Water and Sanitation (EWS) conducts in-plant water sample analysis and some raw water analysis. PLPH conducts the distribution system analyses for compliance with established guidelines and some raw water analyses. The methods used by each of the laboratories will be briefly outlined.

EWS collects and analyzes raw and treated water samples in the Rossdale and E.L. Smith Plants. Total coliform analyses are done by the presumptive MPN procedure in lactose broth at 35°C (draft methods manual, City of Edmonton, M. Gordon). Standard plate counts (SPC) are done by pour plates on tryptone glucose yeast medium incubated at 35°C for 48 ± 3h. It is noted that in the sample collection portion of the most recent draft bacterial methods manual (section 3.24.5) the person collecting the sample is directed as follows: "After 5 minutes of purging the sample line, the bottle is filled to the neck and shaken vigorously 25 times." This is not the correct procedure. The sample must not be shaken at the time of sampling but rather at the time of analysis within the laboratories (APHA, 1985). It is imperative that all sample collection personnel be aware of this fact. The technologists conducting these studies are not professionally qualified in this specialty and must conduct many other routine chemical analyses.

The PLPH uses m-Endo LES agar with Gelman membrane filters for TC analysis. They are incubated at 35°C for 24h. FC are performed on m-FC media with Gelman membrane filters incubated at 44.5 ± 0.2°C for 24 h. SPC are conducted with the pour plate method. Laboratory technicians are fully trained in these methods which comprise the majority of their workload (i.e. they are specialized in this area). They are under the supervision of a qualified professional microbiologist.

3.2.1.2. Assess the adequacy of actual practice against published recommended practice and good microbiological practice.

All of the methods described are basically acceptable under Guidelines for Drinking Water Quality (which specifies Standard Methods for the Examination of Water and Wastewater for methodology). In actual practice, however, the membrane filtration (MF) methods for coliforms, used by PLPH, are more commonly used than the MPN methods, and are considered to give better precision and accuracy. Furthermore, positive presumptive MPN tests should be confirmed by further tests (APHA, 1985) which is not done by the EWS laboratories. It would be better for comparative purposes if all tests were done by the MF method to give comparable results, with increased precision. The method has the added advantage that larger samples of water can be processed, giving a greater chance of finding the organism.

For the standard plate counts (more recently called heterotrophic plate counts - HPC), both laboratories use the pour plate method. This method, while acceptable, is the poorest in recovery of bacteria. It is suggested that spread plates or a MF method be tested with a view to replacing the pour-plate method (all described in APHA, 1985).

3.2.1.3 Provide estimates of the reliability and confidence which can be placed on existing data for all bacterial parameters currently being measured.

Since no data are available on check-samples for precision and accuracy, no statistical analysis can be presented. However, since all the methods (with the exception of the MPN) are accepted methods with good precision and accuracy, most of the data on TC, FC and SPC can be accepted as valid. MPN values are notoriously imprecise with large confidence intervals, but are nevertheless valuable in proving the absence of TC. Further verification of data can be made by comparing TC/FC ratios and SPC/TC ratios. Experience with raw water data can usually permit one to flag data which appear suspect. As an example, Table 5.9 of raw water bacteriology from the Rossdale Plant (City of Edmonton, 1984) mean data for TC are 6000/L while SPC are 4200/L. Obviously since TC are a subset of SPC, this does not make sense. Normally SPC are 10^1 to perhaps 10^4 times greater than TC in raw water. It may be that the pour plate method for SPC is too stressful for the bacteria (thermal shock during mixing and medium too rich). All the raw water data for SPC in this table (and Table 5.10 for E.L. Smith) appear to be low in relation to TC and FC data.

The chronic under-reporting of SPCs in the distribution system tends to minimize the concern for bacterial loads and potential bacterial regrowth in the water. SPC bacteria in the distribution system are significant for the following reasons:

1. some are opportunistic pathogens that can infect susceptible persons;
2. when present at greater than about 500,000/L they interfere with the total coliform test, by making it less sensitive.
3. contributes to the organic load that coats pipes and increases demand for disinfectants;
4. some may be coliforms which can cause positive total coliform tests.

3.2.1.4 Recommend any specific improvements in bacterial monitoring protocol which are warranted.

As a result of the above discussions, the following recommendations are made:

1. All TC and FC analyses should be done by MF methods, using a brand of membrane filter that gives high recoveries (e.g. Gelman, Millipore HC).

2. Pour plate methods for SPC should be abandoned because of low sensitivity. The MF method is suggested to replace it since larger volumes can be processed (APHA, 1985).

3. A correct written sampling protocol should be given to all sample collectors. It should include important information on handing and storage of samples.

4. During periods of high raw water pollution when positive TC are found at the plants and the water distribution system, these coliforms should be identified as to species occasionally.

3.2.1.5 Outline any studies which may serve to improve the bacterial monitoring protocol and/or the reliability of bacterial water analyses.

Recommendations are made in the following section.

3.2.1.6 Provide recommendations on state-of-the art quality assurance and quality control for bacterial analyses.

The PLPH already has routine QA/QC built-in to their daily operation. It may be appropriate to incorporate this into a methods manual to ensure that sterility assurance, temperature control, false-positive, false-negative, media life, etc., are always treated in a standard manner. It is recommended that they occasionally (perhaps once per year) engage in comparative studies with another major water laboratory in Western Canada to ensure that precision and accuracy of all methods are acceptable. All water laboratories should follow QA and QC methods outlined in Standard Methods for the Examination of Water and Wastewater (current edition) as well as the older, but still useful references, Handbook for Evaluating Water Bacteriological Laboartories (EPA, 1975) and Microbiological Methods for Monitoring the Environment (EPA, 1978).

For fecal coliform analyses, temperature control of ± 0.2°C is crucial. An NBS-certified thermometer should be installed in the incubator and a written log of daily temperature maintained.

3.2.1.7 Critically evaluate the bacteriological capability of the Edmonton Water and Sanitation laboratories and make recommendations on manpower and equipment needs.

The Edmonton Water and Sanitation laboratories are minimally capable of conducting useful microbiological analyses. The laboratories are primarily devoted to chemical analyses and have the minimum amount of equipment and staff to conduct microbiological studies. While data are generated that are used in process control, there does not appear to be a rigorous program of QA/AC to validate their information. It is not known whether or not all batches of media are tested with strains of bacteria that should yield positive or negative results; such testing is essential to ensure that the methods work.

Furthermore, split or duplicate samples should be taken to ensure comparability with PLPH, which should serve as their reference laboratory. Their draft methods manual should be completed and should include necessary QA/QC methods.

An assessment should be made of the need for bacterial testing within the water treatment plants. If it is decided that it is essential to maintain in-plant microbiological analyses, it must be ensured that a professional microbiologist conduct or supervise the analyses, that properly trained microbiology technologists conduct the analyses, and that at least 10% of analyses are devoted to QA/QC. Unless the data are valid and capable of close scrutiny, they are useless and may be misleading for control purposes. In such a case the PLPH should do all analyses. An effective reporting mechanism could be set up to allow quick reporting of positive samples so as to allow corrective measure to be taken.

5.4.1 Document Relationship of Waterborne Microorganisms to Disease

5.4.1.1 Review the microbiological monitoring data for Edmonton for the past 3 years.

The microbiological data reviewed included the following:

- fecal coliform (FC) and fecal streptococci (FS) data of the raw water obtained from the Hrudey study in March 1986.

- FC, TC and standard plate counts (SPC) of raw water and treated water from the 1984 Annual Report (City of Edmonton, 1984).

- summary graphs of raw water intake TC and FC from 1980-1985.

- Giardia sampling results from August 1983 - May 9, 1984, conducted by the Provincial Laboratory of Public Health (PLPH).

RAW WATER

The raw water source, the North Saskatchewan River is subject to agricultural runoff, wild animal excreta and after entering the city of Edmonton, to urban runoff and its various constituents. The E.L. Smith Plant, by virtue of its upstream location receives raw water considerably better in microbiological quality than the downstream Rossdale Plant. Peak FC concentrations of about 5000/L are present in the intake at Rossdale and about 100/L at E.L. Smith (Figure 1) Peak and TC values of up to 40,000/L at Rossdale and 15,000/L at E.L. Smith occur (Figure 2). The reported geometric means of SPC bacteria range from 25,000 to 220,000/L at Rossdale in 1984 to 24,000 to 200,000/L at E.L. Smith.

The recently completed spring, 1986, sampling program reveals a very significant difference in raw water qualities of the two plants, mainly due to spring run-off (Figure 3,4).

The FC load at the Rossdale intake was as high as 40,000/L while E.L. Smith was only as high as 2,500/L. A major factor was obviously raw water pollution by the Groat Sewer bank discharge which directly impacts the Rossdale bank intake and contained up to 10^6 FC/L. FC/FS ratios (Figure 5) were ca. 0.6 over the study (range ca. 0 to 4) in the Groat Sewer effluent. This suggests pollution by non-human sources (a ratio of greater than 4.1 is indicative of human wastes and ratio less than 0.7 is considered to be non-human waste, e.g. animals, vegetation, insects, soils). It is recognized that there are many reasons why the FC/FS ratio is not always a reliable index of pollution source.

In summary, both water treatment plants are subject to intermittent fecal and other pollution, largely from non-human sources. At the peak of water contamination, the Rossdale plant requires auxiliary treatment (pre-chlorination or pre-sedimentation, or their equivalent, and post-chlorination) in addition to complete treatment (consisting of coagulation-flocculation, sedimentation, filtration and disinfection) according to the raw water quality classification of the Guidelines for Canadian Drinking Water Quality 1978 (Health and Welfare Canada, 1979). The E.L. Smith plant requires complete treatment by this classification.

TREATED WATER

Treated water is analyzed by City of Edmonton personnel at the treatment plants, as well as by the PLPH. In the 1984 report (City of Edmonton, 1984, Table 5.6) daily Most Probable Number TC analyses are reported. Most samples in the year were negative, but during the spring of 1984 (March and April), 10-23% of samples were positive for TC. SPC values were quite low, with < 1000/L as an annual mean (City of Edmonton, 1984, Table 5.7). However, in view of the discrepancies with the SPC counts (noted in section 3.2.1.3) and the inadequacy of pour-plate methods, these very low values may not be reliable.

Water in water treatment reservoirs throughout the city was essentially free of organisms, with both SPC and TC below the level of detection in reported average results (City of Edmonton, 1984, Table 5.3).

The PLPH data on the bacterial quality of the water in the distribution system during 1984 and 1985 are summarized in Table 1. When the doubtful (contain TC), unsatisfactory (contain FC) and confluent (growth of bacteria all over the plates) are combined, there were 0.9% contaminated samples in 1984 and 1.6% contaminated samples in 1985. It is of interest to note that in 1984, most of the contaminated samples appeared in the distribution system in January through May, which includes the periods when the positive TC counts were found in the treatment plants (see above).

The presence of TC organisms in the treatment plants at some time during the year suggests that less than adequate disinfection is achieved. Fortunately, additional contact time (although minimal from the Rossdale plant) after leaving the plant contributes to better bacterial quality in the distribution system. The low percentage of positive TC samples shows that the finished water at the tap meets Canadian guidelines (Health and Welfare Canada 1985).

The data for Giardia analysis indicate that cysts of this organism were not found in microscopic examination of up to several thousand litres of treated water from E.L. Smith and Rossdale in the period from August 8, 1983 to May 9, 1984 (Memo from Scot McClure to Steven Hrudey and attached data). This is reassuring, but it is also important to know how much Giardia, if any, is present in the raw water. Some testing carried after the Giardia outbreak in Edmonton, between October 1982 and April 1983, never resulted in its detection in either raw or finished water (Collier and Macdonald, 1983, Walker et al., 1986) although not much weight can be put on these data because the team had little experience with the methods at that time.

5.4.1.2 Interpret the results of the monitoring with regard to the likelihood of waterborne disease transmission.

In interpreting the results of the monitoring studies described above, only bacteria and protozoan (Giardia) cysts will be considered in detail. Viruses will be addressed by another consultant in this study. However, before an interpretation of the data is attempted, a brief discussion of the disinfection methods used is required.

Two disinfection steps are employed in the water treatment train that contribute to the microbiological safety of the finished water: chlorine dioxide and chloramination. Chlorine dioxide is an excellent disinfectant for bacteria and viruses, and probably protozoan cysts as well (although little information is available). It is considered to be about 3 times as effective at pH 9 as pH 7 (Benarde *et al.*, 1965, Cronier *et al.*, 1977), so is well suited to the current alkaline operating pH of these plants (and was probably adequate even while raw water was dosed without prior pH adjustment). Chloramines, on the other hand, are relatively poor disinfectants that may only be capable of preventing regrowth problems within the distribution treatment system (Walker *et al.* 1986), and are not generally considered appropriate for primary disinfection of the water, particularly when contact times are short. Thus if chloramination is used as the post-treatment step, a reliable and effective primary disinfectant and an adequate contact time must be provided. Furthermore, the disinfectant residual must not cause an undue risk of toxicity to humans. While it is understood that taste and odour problems are important from a public acceptance viewpoint, adequate disinfection must be the primary objective of water treatment.

Although chlorine dioxide and chloramines are relatively effective disinfectants of *Giardia* cysts, extremely long contact times are required. Normally coagulation-flocculation and filtration are considered the primary barrier against giardiasis transmission from surface water supplies. Good particulate removal and a finished turbidity of 0.2 NTU or less, coupled with adequate disinfection is considered adequate for *Giardia* removal and/or inactivation.

Based on current treatment practices and Giardia analyses, the risk of transmission of giardiasis through Edmonton water is not considered to be high. From a bacteriological point of view, distributed water exceeds all applicable guidelines. However, on occasion total coliforms do escape treatment due to excessive loading of the plants with turbidity, colour bacteria, etc. Such overloads occur during spring and during the summer as well, taxing the capacity of the plants (primarily Rossdale) to produce safe drinking water.

5.4.1.3 Review the water source characteristics and considering any limitations of the indicator organisms, provide recommendations for any additional monitoring or source protection which could substantially reduce the probability of waterborne disease transmission.

The raw water microbiological quality has been described in section 5.4.1.1, above. It is a lightly to moderately polluted raw water source that requires extensive treatment to be considered potable. For raw water analysis fecal coliform and fecal streptococcus (FS) testing is recommended. Total coliform values reveal little about the source or extent of contamination, but may be somewhat useful in measuring treatment effectiveness (since finished water is analyzed for total coliforms. Both FC and FS tests are established sanitary indicators and can provide some information on likely source as well. It is also recommended that Giardia testing be conducted on the raw water as a research tool to determine the seasonality of contamination, if any.

More important than types of organisms to study however, is the question of where and how to survey. It is clear that the bank intake of the Rossdale plant is situated where a probable maximum of contamination occurs. Surveys should, as a minimum, be conducted to locate a better place for a water intake. Ideally an intake source as good as E.L. Smith should be sought, but other less-optimal solutions could also be useful.

In view of the high chlorine dioxide demand during spring runoff and the toxicity of chlorite, alternative disinfection schemes should be considered.

If must be stressed that operators must remain acutely aware that prevention of microbial disease transmission is the primary goal of water treatment. Zealous pursuit of "taste-free" water must not overshadow the need to constantly ensure effective and adequate disinfection.

7.2.2 Evaluation Point-of-use Treatment Options

7.2.2.1 Outline the current choice of options for point-of-use treatment devices currently available in Canada.

Point-of-use water treatment devices can be used in the home in order to overcome a number of common water problems (Tables 2,3). For municipally-treated water with residual taste, odour and chemical problems, the choice can be narrowed down to activated carbon, reverse osmosis and distillation devices

(Tobin, 1984). The uses, advantages and disadvantages of each of these devices will be briefly reviewed.

ACTIVATED CARBON FILTERS

Activated carbon filters are the most common devices in the market due to their wide availability, their uses for taste, odour and chemicals removal, and their relatively low cost. Free available chlorine is almost entirely removed by these filters while some of the total available chlorine, i.e. the combined forms, are left behind. In the same fashion, not all of the organics are removed, with perhaps some of the natural organics in the water being left behind. The US EPA studied the effectiveness of a number of activated carbon filters and reported a large range of organic chemical removals (Bell et al., 1984. THM removals, for example ranged from 4 to 99%, depending on the device tested. Many volatile chemicals can be removed fairly well by many of the filters; chemicals tested included 1,1,1-trichloroethane and trichloroethylene, which have been found in public drinking water and contaminated wells, (spiked at 50 μg/L); carbon tetrachloride, representing a contaminant in chlorine and spills (spiked at 20 μg/L); p-dichlorobenzene (10 μg/L), hexachlorobenzene (10 μg/L) and chlordane (50 μg/L), representing pesticides. Removals of 70-99% were common.

Choramines in water have caused acute hemolytic anemic characterized by Heinz Bodies in dialyzed uremic patients in hospitals using only reverse osmosis to purify the water (Eaton et al., 1973). In hospitals using activated carbon to treat the water, the chloramines were removed and there were no problems among the the dialysis patients.

The relative effectiveness of such devices does not come without a price however. In this case it is the growth of bacteria on the filter medium. Due to the nutritive material present in the water and the bacteria present there as well, there accumulates bacteria and substrate which, under the right temperature conditions, such as overnight stagnation, will grow to fairly high levels (up to 10^9/L) (Tobin et al., 1981). Health and Welfare Canada has established a policy, that has been accepted by the Canadian Water Quality Association and the National Sanitation Foundation in the USA that activated carbon filters should be labeled for use on water that is microbiologically acceptable before treatment. This should eliminate most of the misuses that could give rise to human health problems.

REVERSE OSMOSIS

The science of membrane manufacturing has improved so much in the last few years that it is now possible to operate reverse osmosis devices at tap pressure on city water. This has led to the development of a number of new devices on the market, some of which appear to produce a fairly good water. They are capable in theory of removing a very broad spectrum of inorganic, organic microbiologic and fibrous materials. Cationic species are particularly well removed, but anions such as nitrates are removed relatively inefficiently. They seem to be gaining in popularity. One should be aware that celluose acetate membranes are relatively sensitive to biological and chlorine degradation, but a number of new and more resistant membranes are available. One of the major disadvantages of the devices is the very low efficiency with only about 10% of the water being purified and the rest used to flush the membrane, goes to waste.

DISTILLATION

A number of small stills for the distillation of tapwater, raw water or even salt water, are now on the market. The units consist of a metal or pyrex glass boiler fitted with an immersion heater, an air cooled condenser tube or condensing dome and a collecting reservoir for the distillate. Most home units are designed for counter top use and manual filling of the boiler. Automatic shut-off of the cycle is provided. Larger units may be directly hooked up to a water line.

Because of the temperature of the water and steam in the distillation of water, all harmful pathogens should be inactivated as well as physically removed. The distilled water receiving container, however, may become contaminated with bacteria such as Pseudomonas after a certain period of time.

Although distillation is an accepted disinfection, it is sold even more commonly for chemicals removal. A great deal of promotional literature regarding hazards and effects of poor water supplies on health and the benefits of drinking "pure" distilled water is associated with the sales of these devices. It is often implied that no matter how polluted the water is, most, if not all, impurities are left behind on distillation and that the condensate is essentially safe, pure and wholesome. It should be recognized that many volatile organics will be carried over into the condensate and, depending on the volume collected, may actually concentrate in the treated water. Thus, efficacy data on claims made or implied for removal of organics such as chloroform, pesticides, herbicides, etc. should be backed by test data. In other respects, such as mineral removal, there is no reason why an excellent quality water should not result.

EVALUATIONS

There are a number of organizations that do evaluations of point-of-use water treatment devices of interest to the Canadian situation. They include Health and Welfare Canada, National Sanitation Foundation, Consumer and Corporate Affairs Canada, the United States Environmental Protection Agency or EPA, and the Canadian Water Quality Association. Health and Welfare Canada evaluates and tests devices as time and funding permits and provides advice to consumers, other departments, governments and agencies. The National Sanitation Foundation (P.O. Box 1468, Ann Arbor, Michigan 48106) is developing test protocols for all the types of claims made for water treatment devices. Currently they have standards relating to health-related and aesthetic-related claims for point-of-use devices. They are starting to develop standards for testing disinfection devices such as UV devices. It is possible to obtain from them a full listing of devices that have been tested and found effective in the removal of a number of contaminants. Health and Welfare Canada has been collaborating with the NSF in this effort and currently has members on their working groups and on the Council of Public Health Consultants.

CONCLUSION

In conclusion, any of these three types of devices may be used in the home to remove taste and odour from water. Activated carbon, alone or combined with reverse osmosis or distillation, is the type most likely to provide a significant aesthetic and chemical improvement. Such treatment, however, probably results in an increase in the bacterial concentrations in water. The ingestion of such treated water by healthy adults is not likely to result in any significant health hazard.

REFERENCES

APHA, 1985. Standard Method for the examination of water and wastewater. 16th Edition., APHA, Washington, D.C.

Bell, F.A., Jr. Perry, D.L., Smith, J.K. and Lynch, S.C. Studies on home water treatment systems. J. Amer. Water Works Assoc. 76, 126-130.

Bernard, M.A., Israel, B.M., Olivieri, V.O., and Granstrom, M.L. 1965. Efficiency of chlorine dioxide as a bactericide. Appl. Microbiol. 13, 776-780.

City of Edmonton. 1984. Water Treatment Plants - 1984 Annual Report. Water and Sanitation Department. Water Treatment Section.

Collier, M.K. and Macdonald, P. 1983. Giardiasis - Edmonton Alberta. Canada Diseases Weekly Report 9, 189-192.

Cronier, S., Scarpino, P.V., Zink, M.L. and Hoff, J.C. 1977. Destruction by chlorine dioxide of viruses and bacteria in water. Abstr. Ann. Meeting Amer. Soc. Microbiol. 1977, 238.

Eaton, J.W. Kolpin, C.F. and Swofford, H.S. 1973. Chlorinated urban water a cause of dialysis- induced hemolytic aneunia. Science 181, 464.

EPA, 1975. Handbook for evaluating water bacteriological laboratories. EPA-670/9-75-006. United, States Environmental Protection Agency.

EPA, 1978. Microbiological methods for monitoring the environment. Water and Wastes. EPA-600/8-78-017. United States Environmental Protection Agency.

Health and Welfare Canada. 1979. Guidelines for Canadian Drinking Water Quality 1978.

Tobin, R.S., Smith, D.K. and Lindsay, J.A. 1981. Effects of activated carbon and bacteriostatic filters on microbiological quality of drinking water. Appl. Environ. Microbiol. 41, 646-657.

Tobin, R.S. 1984. Water treatment for the home or cottage. Can. J. Public Health 75, 79-82.

Walker, G.S., Lee, F.P. and Aieta, E.M. 1986. Chlorine dioxide for taste and odour control. J. Amer. Water Works Assoc. 78, 84-93.

Table 1. Edmonton water samples analyzed by the PLPH in 1984 and 1985

MONTH	1984 S	1984 D	1984 U	1984 C	1985 S	1985 D	1985 U	1985 C
January	262	4	-	1	328	1	-	-
February	285	2	1	-	293	2	-	-
March	276	2	3	-	332	2	-	2
April	259	1	-	-	332	-	-	-
May	312	8	-	-	375	3	-	-
June	286	2	-	-	366	4	-	-
July	267	-	-	-	327	6	-	-
August	272	2	-	-	309	6	-	-
September	254	1	1	-	302	8	-	-
October	254	-	-	-	304	6	2	1
November	296	-	-	-	314	8	-	-
December	211	-	-	-	210	8	1	-
TOTALS	3234	22	5	1	3792	54	3	3

Legend: S Satisfactory, D Doubtful, U Unsatisfactory, C Confluent

Table 2. Devices for disinfection of water

Type	Advantages	Limitations	Special Requirements
Ceramic Candles	Inexpensive, simple	May not be effective against viruses	
Chlorination	Proven technology	May require additional filters to remove protozoan cysts	Residual test kit, electricity
Distillation	Effective against all pathogens	Batch system - water may not be available when required	Electricity
Iodination	Inexpensive	May require additional filters to remove cysts. Possible physiological effects of iodine	Residual test kit
Ozonation	Low operating cost	May require additional filters to remove cysts. Iron, sulphur and manganese must not be excessive	Electricity
Ultraviolet Light	Uses no chemicals	May require additional filters to remove cysts	Electricity, UV monitor

Table 3. Devices for taste, odour and chemical removal

Type	Advantages	Limitations	Special Requirements
Activated Carbon	Removes taste, odour, organics, chlorine, low levels of hydrogen sulphide	Should be used on microbiologically safe water	Cartridges should be changed regularly
Chlorination	Iron, manganese, hydrogen sulphide removal		Electricity, filter to remove precipitates
Distillation	Removes inorganic salts, some organic chemicals, particles, hydrogen sulphide	May concentrate some organic chemicals	Electricity
Green Sand Filters	Iron, manganese, hydrogen sulphide removal	May clog or reduce effectiveness due to bacterial growth	Backwash and regenerate regularly, replace media
Particulate Filters	Turbidity, particle removal		
Reverse Osmosis	Inorganic salts removal	Susceptible to clogging	Adequate water pressure, flush
Softeners	Hardness (calcium, magnesium) removal	Elevates sodium content; possible bacterial growth	Usually require electricity

Fig.1 Raw Water Intake Fecal Coliforms, 1980-85

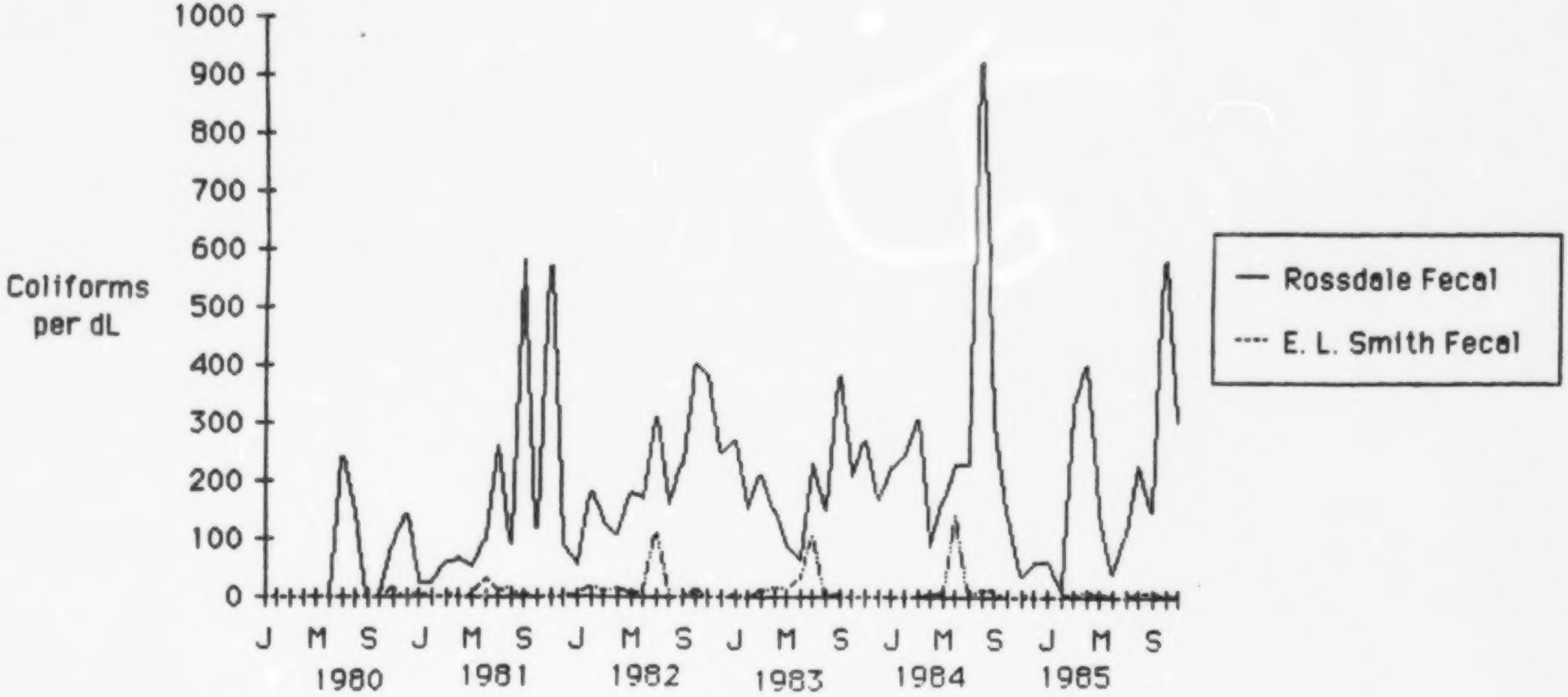


Fig.2. Raw Water Intake Total Coliforms, 1980-85

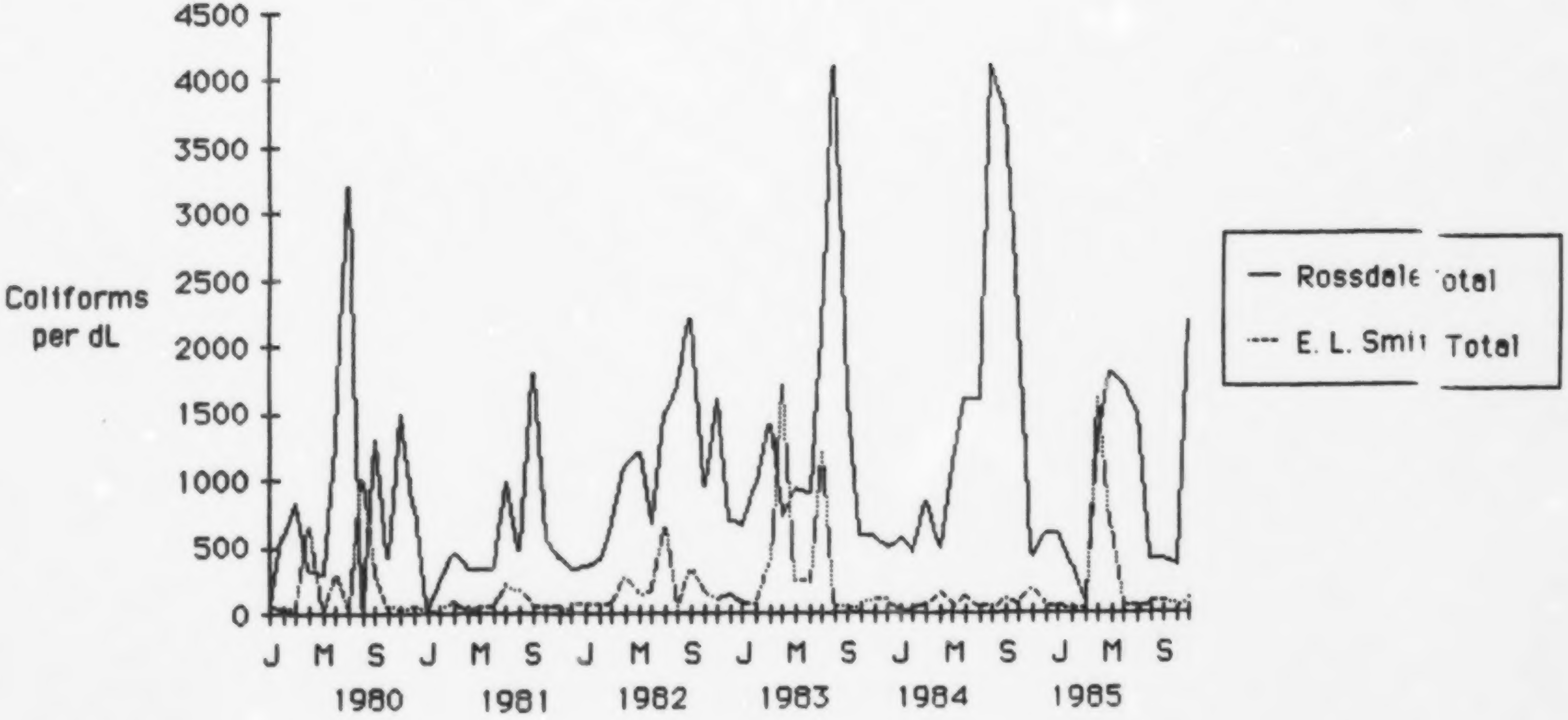


Figure 5. Groat Sewer Outfall

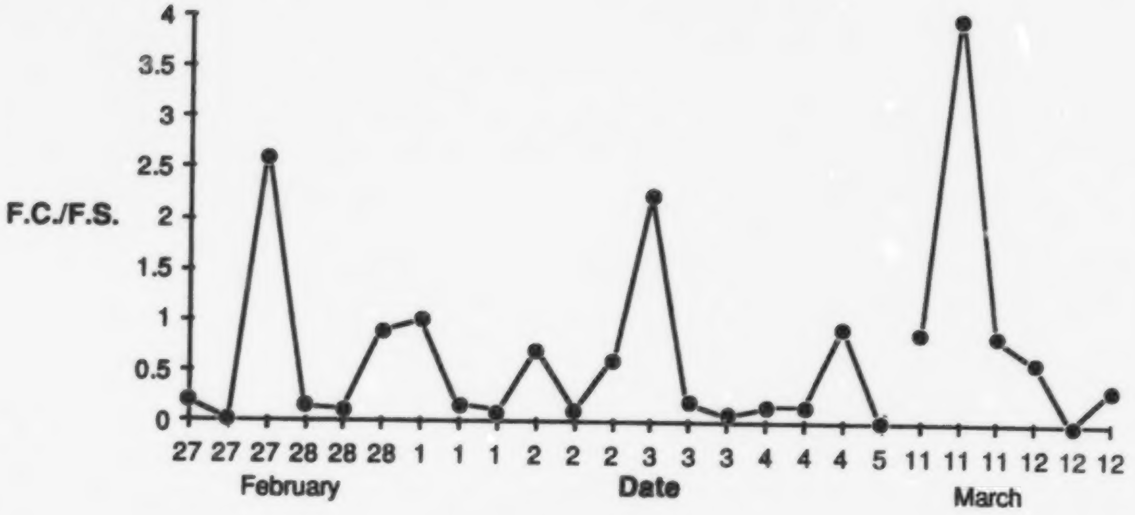


Figure 4. North Saskatchewan River (at Smith and Rossdale Intakes)

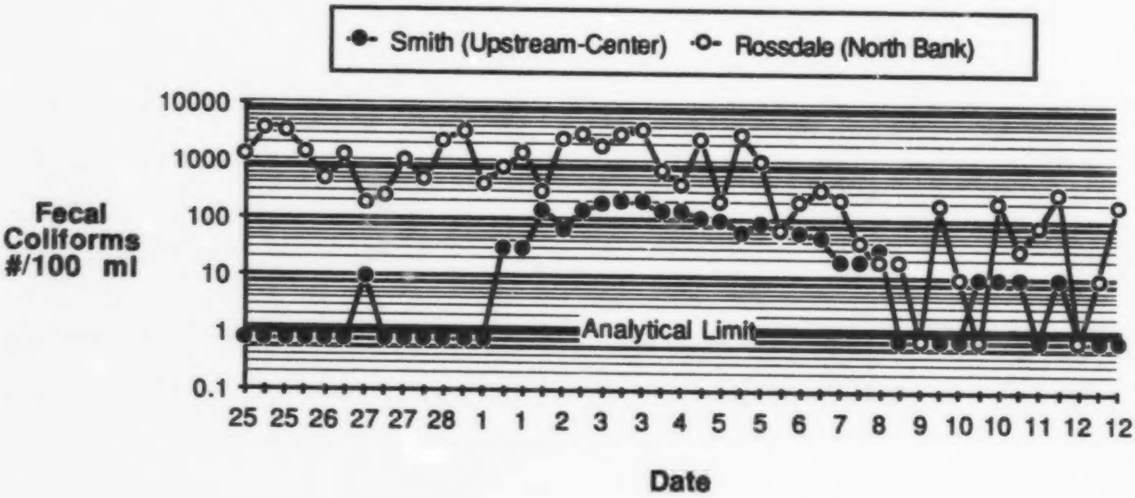
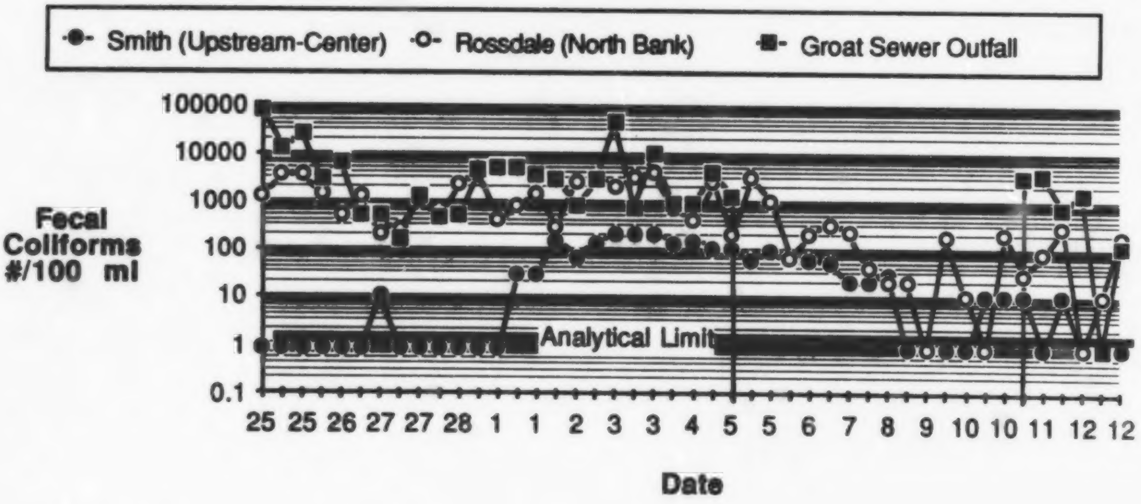


Figure 3. North Saskatchewan River (at Smith and Rossdale Intakes) and Groat Sewer outfall



JAMES M. MONTGOMERY, CONSULTING ENGINEERS, INC.
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April 29, 1986

Dr. Steve E. Hrudey
Professor of Civil Engineering
Dept. of Civil Engineering
The University of Alberta
Edmonton, Alberta
T6G 2G7

Dear Steve:

Enclosed is a short report summarizing our observations, analyses, and recommendations on the City of Edmonton taste and odor(T&O) problems. In spite of the complexity of the problem there are promising short term measures such as filter media replacement with granular activated carbon and a number of potential long term solutions.

Please note that these comments and recommendations are only based on limited knowledge gained from the two-day on-site visit at the end of last month and because only a few days time have been allotted to complete this work the discussion is brief and literature references have not been provided. General recommendations made in this report should be considered tentative only; a full engineering evaluation should be made prior to implementation.

The taste and odor problem at Edmonton is multi-faceted with a number of contributing factors. Although each of the elements of the problem are probably not entirely unique, the combination of all these factors in the Edmonton system does present a unique situation based on experience with U.S. water purveyors. The factors which appear to be contributing to the taste and odor problem are as follows:

Naturally occurring humic and fulvic material

Natural metabolic products of microbiota

Synthetic organic compounds

Disinfection process byproducts

Short term measures which could be taken to alleviate the situation would be 1) optimize the treatment process to remove taste and odor compounds by implementing intensive coagulation and adding disinfectant only after as much T&O contributing material has been removed and 2) replacing the existing anthracite portion of the dual filter media with granular activated carbon.

Long term solutions which should be investigated include 1) ozonation, 2) granular activated carbon contactors, and 3) improved control over sources of T&O and watershed management.

Taste and odor problems are often some of the most difficult problems faced by water treatment engineers and scientists. It should be recognized that if the City wants a long term solution to the T&O problem there will have to be a very significant investment in labor, monitoring, analytical equipment, and plant modifications to be uniformly successful in overcoming this problem. Water purveyors in the U.S., Europe, Japan, and Australia have had the most success in handling tough T&O problems only when research, analytical, water process, and engineering resources have been applied to the problem.

A final comment on one related issue for further review by those conducting the health effects aspect of the Edmonton drinking water assessment program. The high levels of purgeable hydrocarbons found in the Rossdale influent water (for example see the data from the last week of February 1986) warrant further investigation. Gasoline from the City washing into the storm drains has been proposed as the source of these materials. If this is the case a component of these purgeable hydrocarbons may be benzene, a known human carcinogen. This is not unlikely considering that benzene is a percentage level component of some gasolines. Of course there will be losses due to volatilization and if the treatment plants are using powdered activated carbon there will be further removals. Monitoring of raw and treated waters for specific volatile organics with detection limits in the range of the 10-100 nanogram/liter by dynamic headspace/GCMS would provide the information necessary to evaluate this important matter.

It has been a pleasure working on this challenging program. If there are any questions regarding the contents of the attached letter report or we can be of further service to Hrudey and Associates or the City of Edmonton don't hestitate to call.

Sincerely,

Albert R. Trussell

Albert R. Trussell, P.E.
Principal Chemist

ANALYSIS OF TASTE AND ODOR PROBLEM

This section attempts to define what the taste and odor problem is specifically and the potential sources of the problem. The history of the problem, recent problems, and an overview of taste and odor in Edmonton drinking water are presented.

History of the Edmonton Taste and Odor Problem

An understanding of the historical context of a problem is often helpful in fully understanding or solving such a problem. Probably a great deal of information could be gathered from City records and local libraries. A bit of information was gathered talking to the staff of the Department of Water and Sanitation. Data on early treatment practices is sketchy but apparently there was no disinfection of the public water supply prior to the 1920-1930 era. Edmontonians probably simply accepted the tastes and odors that came along with the Spring runoff. Disinfection with chloramines was instigated somewhere around 1920-1930 and Edmontonians put up with a chlorinous-musty odor each year when the Spring runoff occurred. In the last two decades the City experienced tremendous growth due to the booming oil industry and a new water quality problem became significant-runoff of hydrocarbons from urban areas into the raw water supply. Then in 1982 the water disinfection practice was changed to include chlorine dioxide and this introduced an additional problem- disinfection byproducts with a spicy chemical odor.

Recent Taste and Odor Problems

On March 1-6, 1986 both the E.L. Smith and Rossdale water treatment plants experienced severe problems with a taste and odor episode. In the previous week there had been record high temperatures and high levels of hydrocarbons caused by City runoff had been successfully treated at the Rossdale plants by adsorption on powdered activated carbon and oxidation with chlorine dioxide. The "upstream runoff" during the first week of March was a different case and was not successfully treated.

I assessed the aroma and flavor of water samples that had been stored under refrigeration since the T&O incident when I visited Edmonton. At that time the samples were about three weeks old. The samples undoubtedly underwent some microbiological degradation during this period and it can only be presumed that the same odor notes were present based on the observation of plant staff who felt the water "still had the same smell." Remembering the particular odor note of the original incident they may have been able to pick it out in spite of a change in relative intensity with other notes that were now present. Recognizing these limitations these are the results of the assessment:

RAW WATER E. L. SMITH- MARCH 5, 1986

	Description	Intensity
AROMA:	musty/earthy	moderate
	grassy	moderate
FLAVOR:	earthy	strong
	grassy	strong

TREATED WATER E. L. SMITH DISTRIBUTION SYSTEM- MARCH 3, 1986

AROMA:	sweet	slight to moderate
	spicy chemical	moderate
FLAVOR:	spicy chemical	moderate to strong
	chemical aftertaste	moderate to strong
	bitter	slight to moderate

If these samples were representative of the original taste and odor episode it seems clear that the treatment was modifying the original content to produce a new class of T&O compounds.

Water samples from current operations were also assessed for their flavor profile:

E.L. SMITH FILTERED WATER- MARCH 26, 1986

AROMA:	none	
FLAVOR:	earthy/musty	slight

E.L. SMITH CLEARWELL WATER- MARCH 26, 196o

AROMA:	grassy	slight
	chlorinous	slight
FLAVOR:	earthy/musty	slight
	bitter	moderate

It is possible that the additional contact time with chlorine dioxide experienced by the clearwell water is reponsible for the additional odor notes and bitter aftertaste.

When visiting the Rossdale plant the next day there was not sufficient time to bring samples to room temperature for an adequate flavor profile analysis. However the "sniffer" device that was set up to heat and spray the raw water was sampled. It had a moderate to strong earthy odor characteristic of geosmin; I would venture to say based on experience in lab analyses, and flavor profile panels that the concentration of geosmin (or some very similar homologue) in the raw water was in the range of 5-30 nanograms/liter.

Overview of Taste and Odor Problem

Based on the site visit, discussions with Water and Sanitation staff, and review of historical information that we were provided the T&O problem appears to be three-fold: 1) Natural upstream aquatic humus and other microbiological byproducts, 2) Synthetic organics from urban/industrial runoff, and 3) Disinfection byproducts produced during the water treatment process.

In the Spring when the snow is melting off large slugs of organic material are apparently leached as muskeg bogs overflow with snowmelt. Although there is always a background level of aquatic humus which enters the Saskatchewan River and is measured as Total Organic Carbon (TOC), during snowmelt events the quantity of organic material can increase 3-4 fold as evidenced by the data collected during the first few days of March this year. Aquatic humus itself is a condensation product of microbiological activity on decaying vegetation. It is likely that other microbiological byproduct compounds are diluted into the upstream water along with the humic material. The bogs and sloughs undoubtedly contain actinomycetes in their mats of decaying vegetable matter. We were told that the bogs often contain several feet of water over peat moss and algae growth can be abundant in warmer months. Algal material and exudates that are frozen in the Fall as the bogs freeze over may be suddenly released in springtime thaws. Actinomycetes and certain strains of blue-green algae are known producers of geosmin and methylisoborneol. These compounds are classic microbiotic byproducts which have been determined to be the cause of earthy and musty T&O problems in water supplies on several continents. It is suspected that these compounds(or some very similar homologues) are present in the Saskatchewan based on the flavor profile assessments described above and the consistent use of the the term "musty" by Edmontonians to describe the Spring runoff water. This is all speculation at this point and should be confirmed by monitoring as discussed later in this letter report.

Synthetic organic compounds are also a part of the overall T&O problem. Storm drain runoffs contain high levels of purgeable and extractable hydrocarbons. These hydrocarbons are undoubtedly the cause of Edmontonians complaining about gasoline or hydrocarbon odors in the drinking water. Although this problem is presumed to be entirely an urban runoff problem affecting the Rossdale plants, future monitoring shouldn't preclude assessing the upstream impact of petroleum industry operations and/or naturally occuring hydrocarbon material that may add a subtle odor note to the raw water at the E.L. Smith plant.

Other compounds such as home and garden pesticides and herbicides may also be present in runoff waters. There are limited industrial, power plant facilities, and agricultural areas upstream of the City. There is insufficient monitoring data available to evaluate these impacts on the synthetic organic load in the Saskatchewan River. A sanitary survey and monitoring program is recommended in the section of this report on **Long Term Solutions**.

An additional category of taste and odor compounds are those produced during the water treatment process. In particular it appears that the use of chlorine dioxide is creating obnoxious tastes and odors when it reacts with organic material from upstream runoff. The flavor profile assessments described above demonstrate the change in odor notes after treatment at the E.L. Smith plant with chlorine dioxide.

Under certain conditions gas chromatographic profile analyses conducted at the Rossdale lab show the presence of two additional volatile organic peaks after the addition of chlorine dioxide. The first of these additional volatiles elutes before chloroform and the second elutes after chloroform. Purgeable organics GCMS lab analyses conducted on treated water samples collected in March 1985 showed the presence 2-methyl propanal, 3-methyl butanal, and 2-methylbutanal. Comparing chromatograms leads to a very tentative conclusion that the first peak might be 2-methyl propanal and the second peak might be the methylbutanal isomers. The retention time slots are comparable and C4-C5 aldehydes should respond well on the GC with the flame ionization detector that is used in the Rossdale lab. Previous studies and sampling/analysis of water treatment facilities at Montgomery Laboratories have shown the presence of C5-C12 aldehydes (including 3-methyl butanal) in waters treated with chlorine and ozone and a recent literature review by Dr. Rip Rice identified aldehydes as a byproduct of chlorine dioxide disinfection. Therefore it is not surprising that aldehydes may have been found in treated Edmonton water. The information on the exact compounds that are present and their concentration levels is very limited at this time. Additional monitoring and analyses using properly calibrated techniques and GCMS confirmation of compound identities should be conducted before any firm conclusions are drawn about the exact content of volatile chlorine dioxide byproducts in Edmonton finished water. Low molecular weight carboxylic acids and aromatic acids produced as chlorine dioxide byproducts may also warrant investigation.

SHORT TERM MEASURES TO REDUCE TASTE AND ODOR

There are a number of short term measures that can be taken which may improve water quality with respect to T&O while long term solutions are developed. These include replacement of filter media with granular activated carbon, optimizing the existing treatment process for removal of T&O organics, adjustment of the nitrogen balance through the treatment process, and optimization of selection of powdered activated carbon.

Replacement of the anthracite portion of the filter media with granular activated carbon (GAC) is the most promising of these short term measures and may be a desirable permanent change for reducing T&O complaints. Such a bed of GAC may be effective for several years for reducing the T&O content of the treated water based on the experience of U.S. utilities. Adsorption is the only unit process which is uniformly successful in treating "musty" T&O problems which are typically resistant to oxidation. It would be desirable to find a GAC with an effective size and uniformity coefficient that does not have a negative impact on filter performance and the deeper the GAC bed that can be installed the better. Rough estimates indicate that the Empty Bed Contact Time (EBCT) of a GAC bed that is limited to the 450mm depth of the current anthracite media in the E.L. Smith plant would be 3-4 minutes at typical springtime water treatment plant flowrates. Monitoring the exhaustion of the GAC bed and the issues involved in GAC replacement of filter media are topics in themselves that could be the subject of an entire separate report.

Optimizing the existing treatment process to remove the maximum possible amount of T&O producing organics would involve the following basic steps:

Adding potassium permanganate to the raw water to oxidize organic material as the water enters the plant

Adjusting the pH as necessary and using alum or ferric chloride in conjunction with polycationic polymer to coagulate as much organic material as possible

Adding powdered activated carbon to adsorb as much organic material as possible

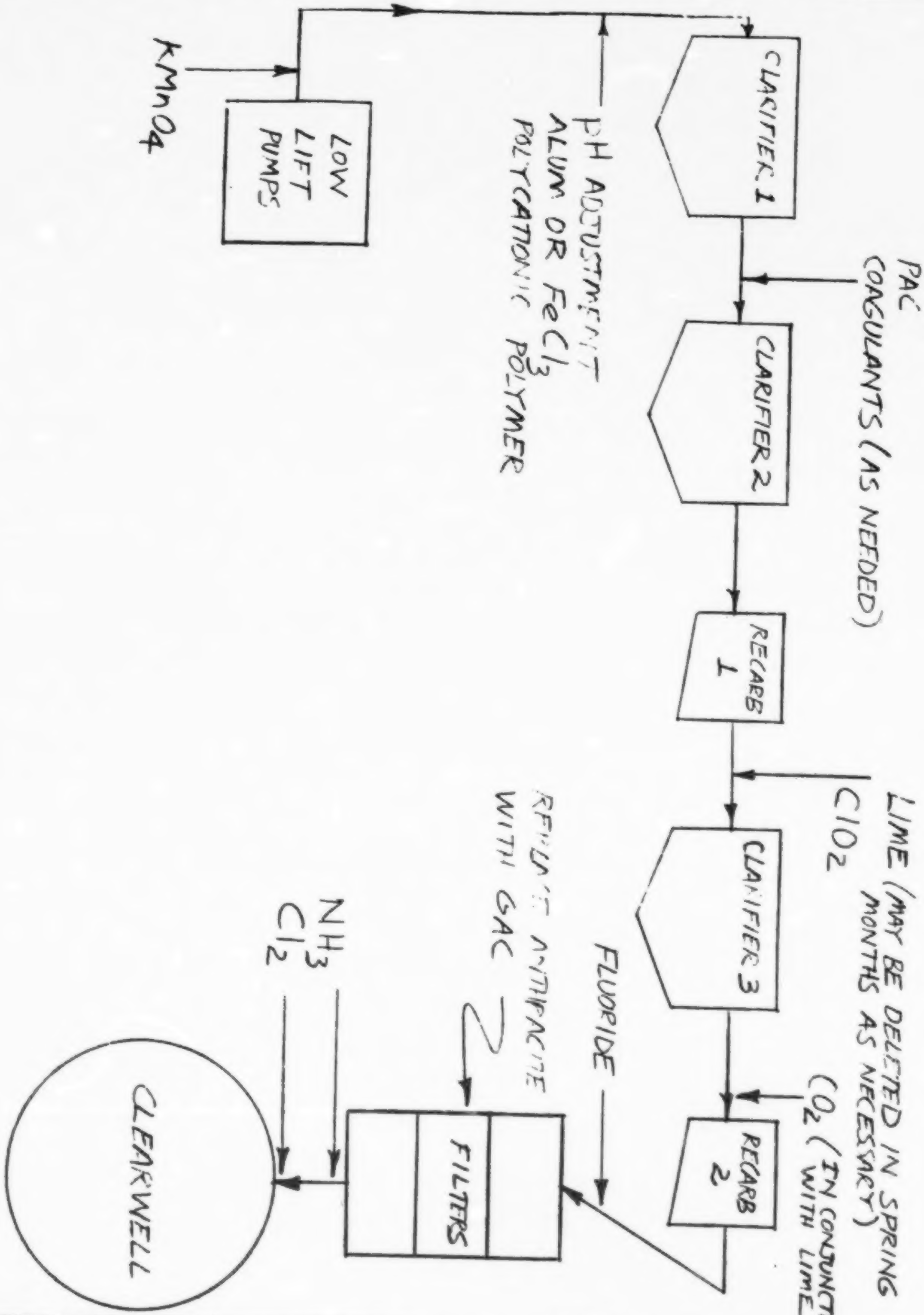
Adding chlorine dioxide as a primary disinfectant and to oxidize any remaining organic material

Rough schematics detailing possible application points for these treatment chemicals are shown in the figures on the following pages. Please note that the figure for the Rossdale plant shows proposed routine operation in the upper half of the figure and proposed Springtime or T&O season operation in the bottom half of the figure. Feasibility and implementation issues associated with these suggested changes cannot be addressed within the limited scope of this report. Suggested changes may not be feasible due to physical constraints or operational difficulties that were not apparent in the cursory site visit. For example although the Saskatchewan River water is not extremely hard, water quality, corrosion, or consumer preference factors may be so strong that temporary periods of operation without lime softening may not be feasible. Any operational changes will have to be considered with critical attention for proper disinfection and maintaining a safe water supply. Such issues must be addressed in future work.

DATE 23 APRIL '86

CLIENT HRUDEY & ASSOCIATES JOB NO. 1826.0010 CALC. BY A.R.T.

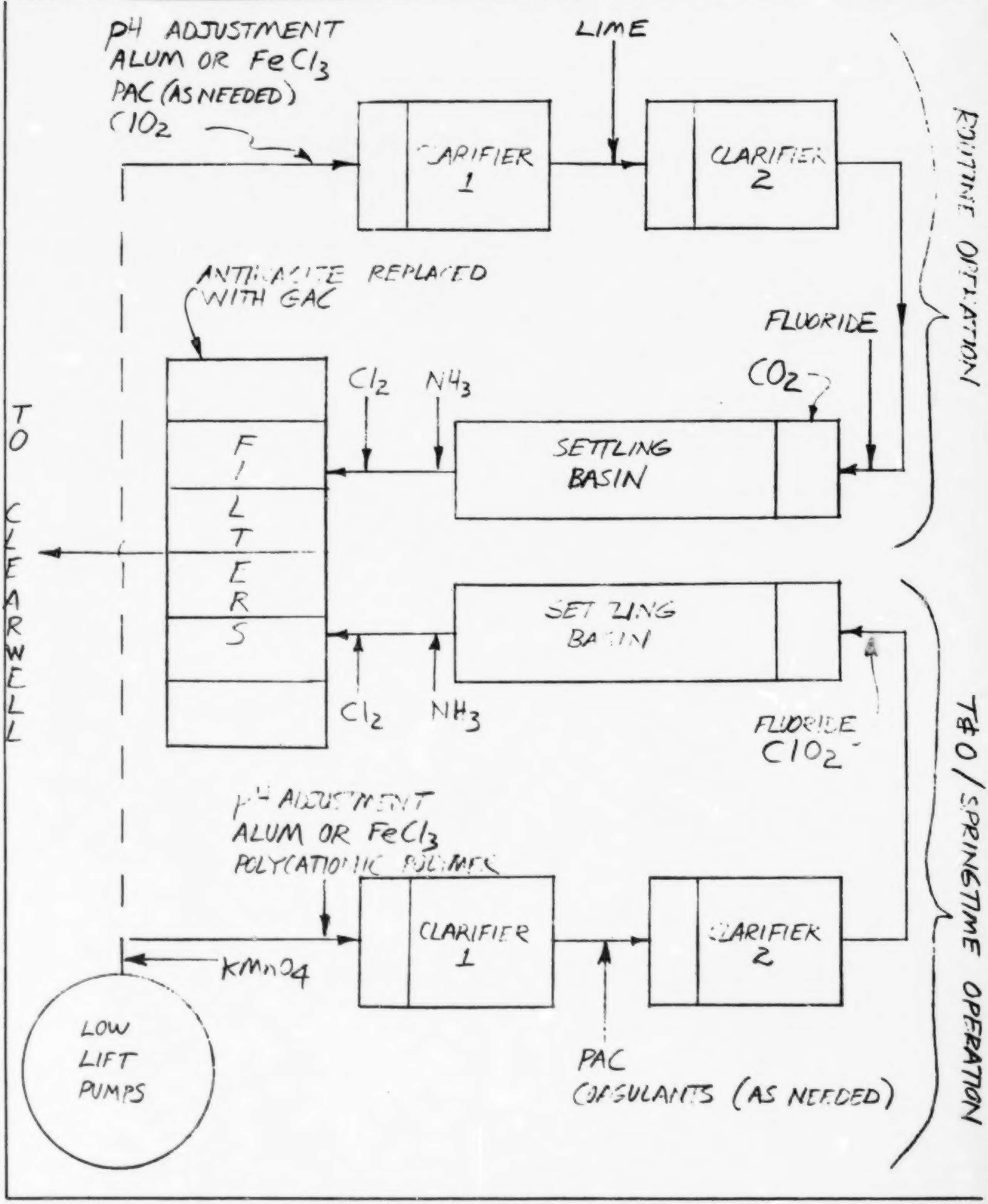
DESCRIPTION PROPOSED OPERATION AT E.L. SMITH PLANT CHECKED BY



DATE 24 APRIL 86

CLIENT HARDY & ASSOCIATES JOB NO. 1826.0010 CALC. BY A.R.T

DESCRIPTION PROPOSED OPERATION AT ROSSDALE 1 & 2 CHECKED BY —



Another issue which should be investigated is the "nitrogen balance" of the water treatment process. Currently ammonia is added without regard for influent water ammonia content and the chlorine:ammonia ratio is not carefully monitored. This could lead to a number of undesirable situations. At chlorine to ammonia ratios higher than 5:1 (on a weight basis) there is the potential for the formation of odorous dichloramine and trichloramine, although the high pH after lime treatment would help to mitigate this effect. If ammonia is overdosed there is a greater potential for reactions which might form organonitrogen compounds, for example nitriles by the reaction of ammonia with aldehyde byproducts of chlorine dioxide treatment. Ammonia overdosing may also lead to a reduced, nitrogen-rich environment in the distribution system which could lead to the possible formation of organonitrogen compounds or biological growths under certain conditions. In any case it would be prudent to carefully monitor and control the chlorine:ammonia ratio at around 3:1 to ensure the formation of a predominantly monochloramine residual.

One last area which could be looked into on the short term would be the selection and use of Powdered Activated Carbon(PAC) with maximum adsorptive capacity for the specific T&O organics found in the Edmonton raw water. Currently PAC is purchased solely on the basis of low bid. PAC from different manufacturers could be rated for iodine number and evaluated in bench scale isotherm tests for specific capacity for removing Edmonton's taste and odor compounds. Although use of a different PAC is not expected to lead to dramatic improvements in T&O compound removal efficiencies, a bulk chemical testing program in general would be valuable for ensuring maximum effectiveness of purchased chemicals.

LONG TERM SOLUTIONS

This section of the report discusses measures which can be taken in the long term to develop a permanent solution to the Edmonton T&O problem. These ideas will require a serious committment of resources and will require implementation over a period of years before they become fully effective. The measures considered as long term solutions are source monitoring and control and treatment with granular activated carbon contactors and/or ozone.

Source Monitoring and Control

A carefully designed monitoring progam carried out over a period of years could develop a database of information that could lead to knowledge of point and non-point sources of T&O compounds or precursors of T&O compounds in Edmonton water.

A sanitary survey of both urban and rural watershed areas for domestic, agricultural, and industrial inputs should be conducted in conjunction with such a monitoring program. As soon as information is developed in the sanitary survey it should be incorporated into the monitoring program. For example when usage patterns of certain pesticides are identified in the survey they should be evaluated for environmental persistence with particular regard for their waterborne fate and the monitoring program should focus on those compounds most likely to actually be present in the raw water. Very little monitoring information was available on pesticides and herbicides in raw or finished water. Certain pesticides have been responsible for T&O problems in U.S. utilities especially after they are oxidized during the treatment process.

Based on the results of the monitoring program and sanitary survey it may be possible to modify or control usage patterns and disposal practices of hydrocarbons and/or other compounds in domestic areas and thereby reduce the quantities released to the Saskatchewan River. The feasibility of this approach versus building a new upstream intake for the Rossdale plants should be evaluated.

Monitoring program results may also offer interesting insights into the sources of the upstream T&O compounds. If results show that water releases from Brazeau Dam are the cause of significant problems an inter-agency program in cooperation with TRANS ALTA may be helpful. Cooperation with other upstream agencies which have an impact on water quality may also be effective. If rapid snowmelt leaching organics from muskeg bogs or sloughs is found to be a significant source of T&O compounds then perhaps an early warning system can be developed based on weather patterns which lead to snowmelt or TOC measurements can be made downriver to predict the influence of the bogs.

Each of the types of analyses recommended for the monitoring program are discussed briefly in the section below. A rough sketch indicating potential monitoring locations is shown in the figure on the following page. Which analyses are selected for exactly which locations will have to be decided based on economic constraints and other feasibility issues. The analyses proposed focus on problems with organics, taste and odor and color. The scope of the program could be expanded to include other water quality aspects such as giardia, virus, asbestos, etc. depending on the needs of the Edmonton Drinking Water Assessment Program.

General Analyses

Total Organic Carbon: a measure of the total quantity of organic material present based on carbon content. Total organic carbon measurements include organic material associated with particulate matter and is best conducted by a high temperature pyrolysis oxidation technique which recovers carbon from larger particles.

Dissolved Organic Carbon: a measure of the soluble fraction of organic material present in a water. This measurement can be conducted by either high temperature pyrolysis or UV-wet chemical oxidation techniques.

Total Organic Halogen: a measure of the total quantity of adsorbable organic compounds containing chlorine, bromine, and iodine expressed on the basis of the Cl which is present. Samples are analyzed by adsorption on activated carbon and pyrolysis with microcoulometric detection.

Organic Fractionation: a measure of the total quantity of organic material present by categorizing it according to fundamental chemical functionalities. By adsorbing and eluting the natural organic materials on XAD resins with a particular protocol the material can be fractionated into six major categories: acidic hydrophobics, basic hydrophobics, neutral hydrophobics, acidic hydrophilics, basic hydrophilics, and neutral hydrophilics. This information can be used to assess percentages of humic and fulvic acids present and the contribution of other classes of organic compounds to the the total organic composition contained in the water.

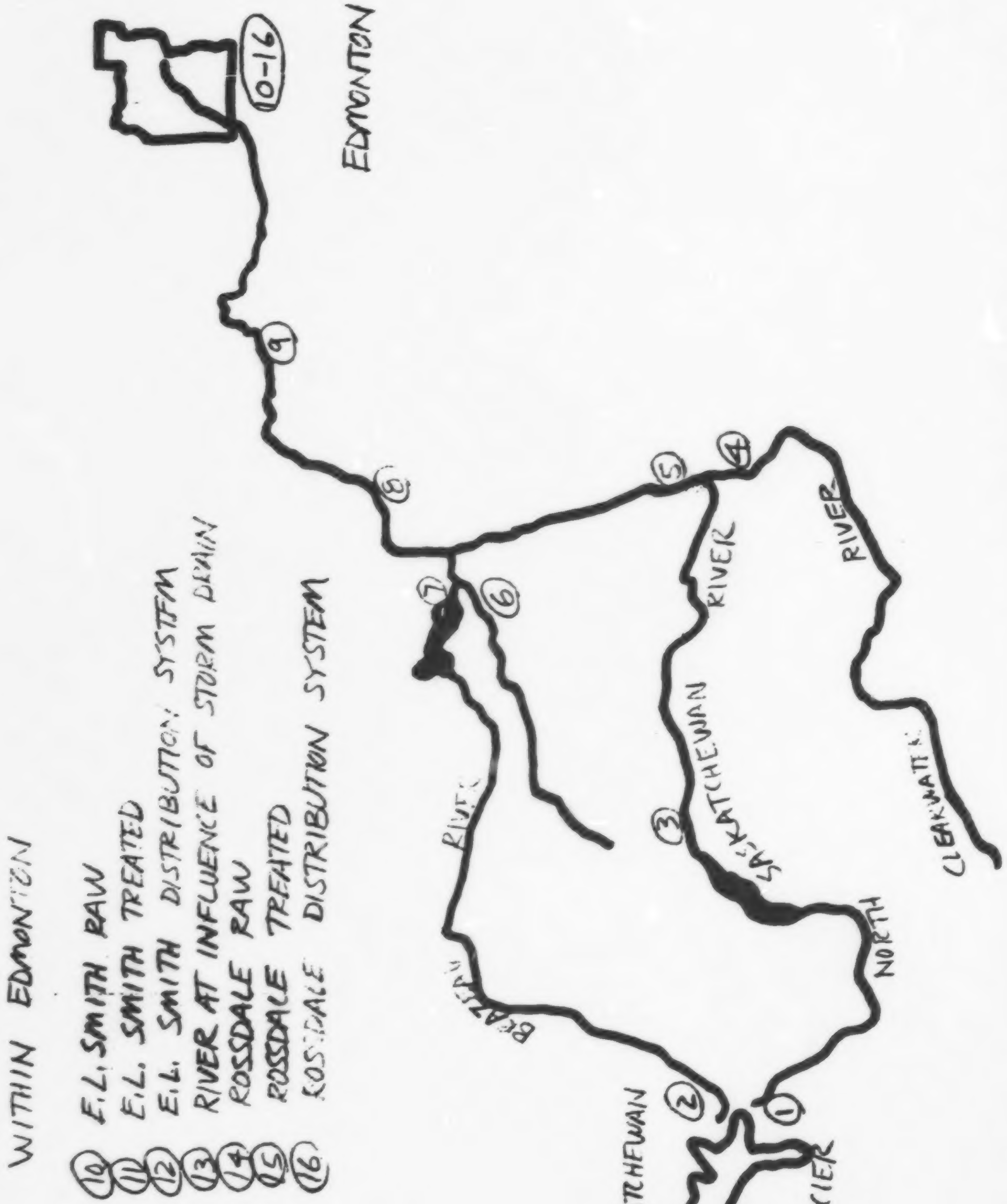
Taste and Odor Analyses

Closed Loop Stripping: semi-volatile organics are stripped from water and adsorbed onto an activated carbon trap. The carbon is then extracted with carbon disulfide which can be injected into a GCMS or GC equipped with flame ionization detector. Instrumental detection limits are in the range of 1-10 ng/L for many compounds. Table I on the following page lists a number of potentially odorous compounds found in raw and finished waters using this technique in the Montgomery Trace Organics Laboratory.

DATE 24 APRIL 86

CLIENT HRUDEY & ASSOCIATES JOB NO. 1826.0010 CALC. BY A.R.T.

DESCRIPTION MONITORING LOCATIONS CHECKED BY —



WITHIN EDMONTON

⑩ E.L. SMITH RAW
⑪ E.L. SMITH TREATED
⑫ E.L. SMITH DISTRIBUTION SYSTEM
⑬ RIVER AT INFLUENCE OF STORM DRAIN
⑭ ROSSDALE RAW
⑮ ROSSDALE TREATED
⑯ ROSSDALE DISTRIBUTION SYSTEM

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JAMES M. MONTGOMERY, CONSULTING ENGINEERS, INC.
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SHEET 1 OF 1

Purge and Trap: volatile organics are stripped from water into a Tenax trap and then the compounds are desorbed and focused at the front end of a capillary column with a cryogenic trap. The capillary column is directly interfaced with a mass spectrometer for analysis. Instrumental detection limits are in the range of 10-500 ng/L depending on exact operational conditions. Detection limits can be further reduced when searching for a known compound by employing Single Ion Monitoring(SIM). The SIM purge-and-trap approach has been successfully employed as a rapid analysis technique for the specific odorants methylisoborneol (MIB) and geosmin. This technique also recovers organic sulfur compounds often implicated in "swampy" T&O problems, volatile synthetic organics, and purgeable hydrocarbons. This purge-and-trap method precisely recovers very volatile compounds which are lost during the carbon disulfide extraction step of closed loop stripping.

Steam Distillation: polar volatile organics are distilled from water and the steam is continuously extracted with pentane in a special apparatus. The final pentane extract can be injected into a GCMS or a GC equipped with flame ionization detector or alternatively the GC effluent can be diverted to a human nose to develop an odorgram profile of constituents in the water. Table II lists a number of compounds found in finished waters using this or similar steam distillation techniques in Montgomery's Trace Organics Laboratory. This method recovers compounds which are not recovered by gas stripping techniques such as purge-and-trap and closed loop stripping.

Sensory Assessment: the flavor profile of raw and finished waters can be evaluated by a panel of 3-5 persons. Raw waters should not be taken into the mouths of panel members unless there is sufficient evidence that treating the samples with sub-micron filtration will remove any microbiological hazard. The flavor profile should include a test of both the aroma and taste of the water with ratings of taste intensity, sensation, quality, and aftertaste. Flavor profile results can be augmented by the more classical threshold odor numbers developed by serial dilution of water samples until no odor is detectable. Results from sensory assessment analyses should be correlated with detailed complaint logs documented for consumer complaints.

TABLE I.
POTENTIALLY ODOROUS COMPOUNDS TENTATIVELY IDENTIFIED IN RAW AND FINISHED WATERS USING CLOSED-LOOP STRIPPING

Compound	Location Found
1-Methyl-4-(1-methylethenyl)cyclohexene	CA, CO
1,3,3-Trimethyl-2-oxabicyclo/2.2.2/octane	CA, CO, VA
1-Methyl-4-(1-methylethyl)-7-oxabicylco/2.2.1/heptane	CA, CO, VA
1,3,3-Trimethyl-2-oxabicyclo/2.2.1/octane	CA
3,5,5-Trimethylbicyclo/4.1.0/heptan-2-one	CA
1,3,3-Trimethylbicyclo/2.2.1/heptan-2-ol	CA, CO
1,3,3-Trimethylbicyclo/2.2.1/heptan-2-one	CA, CO
4-Methyl-1-(1-methylethyl)bicylco/3.1.0/hexan-3-ol	CO
2,6,6-Trimethylbicyclo/3.1.1/hept-2-ene	CO
1-Methyl-4-(1-methylethyl)-3-cyclohexen-1-ol	CO
5-Methyl-2-(1-methylethyl)cyclohexanol	CO
2,6,6-Trimethylbicyclo/3.1.1/hept-2-one	CO
2,2-Dimethyl-3-methylenebicylco/2.2.1/heptane	CO
4,4,6-Trimethylbicyclo/3.1.1/hept-3-en-2-one	CO
Octahydro-3,6,8,8-tetramethylmethanoazulene	CO
Trimethyldisulfide	Aust.
Dimethyldisulfide	Aust., CO, CA

CA = California
CO = Colorado
VA = Virginia
Aust. = Australia

TABLE II.
COMPOUNDS TENTATIVELY IDENTIFIED IN FINISHED WATERS USING STEAM DISTILLATION

Ethanol
Decanal
2-Propanone
6-Methyl-2-heptanone
3-(1-Methylethyl)oxetane
Azulene
2-Methylbutanal
4-Methyl-1-(1-methylethyl) 3-cyclohexen-1-ol
Undecanal
Heptanal
2-Methyl-2-propanone
1,1,2-Trichloro-1,2,2-triflouroethane
2-Methylpropanal
3-Methyl-2-butanone
2,3-Dimethylsuccinonitrile
2-Nitropropane
Benzaldehyde
Methyloxirane
2,2-Methyloxirane
2,3-Butanedione
1,3,5-Cycloheptatriene
Octanal
Chloroacetonitrile
3-Hexanone
Bicyclo/4.2.0/octa-1,3,5-triene
2,2,6-Trimethylcyclohexanone
2,3,3,4-Tetramethylpentene
2,3-Pentanedione
1,2,3-Trimethylcyclohexane
3,5,5-Trimethyl-2-cyclohexen-1-one
4-Methyl-2-propyl-1-pentanol
2-Propenal
Acrylonitrile
2-Methylpropanenitrile
Butanal
2-Methyl-2-butanol
Propanenitrile
2-Butanone
Tetrahydrofuran
Pentanal
2-Hexanone
Hexanal
2-Ethyl-1-hexanol
1-Octanol
Nonanal
Acetonitrile
3,5,5-Trimethyl-2-cyclohexen-1-one
2-Butene
Methyloxirane
3,3-Dimethylhexanal
1-Pentanol
Hexanol
4,4-Dimethyl-2-oxetanone
1,3,5,7-Cyclooctatetraene
1,2,3-Trimethylbenzene
3,3-Dimethylhexanal
Pentafluoro/(pentafluorophenoxy)methyl/-benzene
2-Methoxy-2-methylpropane
1,1-Dichloro-2-propanone
4-Methyl-2,3-pentanedione
Butylacetate
6-Methyl-1-heptanol
4-Methyl-2-propyl-1-pentanol
2,2,6-Trimethylbicyclo-/3.1.1/heptane

Other Organics Analyses

Acidic Extractables: acidic organic compounds can be extracted at low pH and derivitized in situ with the acylation procedure developed by Dr. Coutts. Extracts can be analyzed be GC and GCMS.

Base/neutral Extractables: basic and neutral semi-volatile organics can be extracted at pH 11 and analyzed by GC or GCMS. This analytical fraction will contain the extractable hydrocarbons.

Pesticides/Herbicides: pesticides and herbicides will have to be analyzed in several separate analytical fractions based on chemical classes. Typically analyses are done for the following types of fractions: organochlorine, organophosphorus, triazines, carbamates, chlorophenoxy herbicides, etc. Some compounds such as glyphosate and paraquat may require individual analyses designed specifically for that component.

Microbiological Analyses

Actinomycetes: actinomycetes can be isolated and enumerated on starch casein salt agar medium as specified in **Standard Methods**.

Algae: samples of both planktonic (free-floating) and periphyton (attached) alga should be collected and analyzed. To determine predominant species wet mounts can be initially scanned at low magnification (200X). The specimens can be examined at higher magnifications (500-1000X) using both differential interference contrast and brightfield microscopy for specific keying.

Other: at the time of collection of samples for algae and actinomycetes assays it would also be of interest to note the presence of any macrophytes (macroscopic aquatic plants) that might be rotting and contributing T&O compounds to the raw water.

Other Treatment Options

There are two additional unit processes which are often very effective in removing tastes, odor and color from water. These are ozonation and granular activated carbon post-filtration contactors.

Ozone is the most powerful oxidant used in drinking water treatment and is effective in removing a wide variety of T&O compounds, reducing color levels, and reducing trihalomethane formation potential in many waters. Ozone also improves the coagulation performance when used as a pre-oxidant. However ozone is not particularly effective on the musty/earthy T&O compounds, MIB and geosmin. Ozone must be generated on site and considering the capital and operation and maintenance costs a careful pilot evaluation is recommended prior to selection as an element of the Edmonton water treatment practice.

GAC contactors would typically provide at least 4-5 times the adsorptive capacity as compared to GAC replacement of the anthracite filter media. A typical contactor for T&O removal would probably be designed with a 15 minute empty bed contact time, whereas GAC in the filters would have only 3-4 minutes EBCT depending on plant flowrates. The GAC would probably continue to remove T&O for several years based on European experience, but more frequent replacement or regeneration would be necessary if removal of specific organic compounds was desired. It should be noted that GAC contactors are breeding grounds for nematodes and other types of macroscopic organisms. Therefore the contactors will have to be backwashed at intervals thereby reducing the maximum adsorption efficiency. GAC contactors would be a large investment and besides the ecomonic evaluation of cost/benefit ratio the technical issues such as EBCT, breakthrough volumes, breeding of macroscopic organisms, the ability of mature carbon to handle an accidental chemical spill in the river, etc. should be evaluated in pilot scale studies prior to implementation.

OTHER RECOMMENDATIONS

General Approach

One final area to consider is the overall administrative approach to tackling the taste and odor problem and other water quality/water treatment issues in Edmonton. Water purveyors in the U.S., Europe, and Japan that are successfully overcoming such problems uniformly have channelled resources to the areas of analytical methods development, water process research, pilot studies, and new facilities. Often there is a whole division or department dedicated solely to such work.

Water treatment plant and laboratory staff that we met during our site visit were keenly interested in improving the quality of water treatment and monitoring efforts. Without staffing and resources available to be dedicated to water process development the only option for testing new treatment chemicals and plant modifications has been full plant trials. Although all process changes eventually must be implemented at plant scale this is not the most prudent testing ground for new ideas because of its direct and immediate impact on water consumers. In spite of the limited resources available the existing staff have made significant improvements in plant performance in recent years as evidenced by improved removal of hydrocarbon odors and color.

The City has made large investments over the years in water treatment facilities. The E.L. Smith plant is one of the most beautiful and modern plants that I have toured in recent years. The current upgrade of the Rossdale plants will assist in improving water quality and provide more operational flexibility. The City has provided space and assistance to Dr. Peter Huck in his current state-of-the-art pilot study of the disinfection byproducts in treated Saskatchewan River water. It is evident that the City of Edmonton has shown a continuing interest in providing a safe water supply for Edmontonians. It light of new analytical methodologies and recent developments in the field of water treatment it is recommended that the City augment these efforts by expanding laboratory capabilities and forming a staff unit dedicated to water treatment process development. These capabilities should include a semi-permanent small scale pilot plant unit.

Laboratory Operations

The laboratory staff and facilities will contribute heavily to implementing any of the short or long term solutions discussed above. Currently the staff resources available are utilized almost entirely for routine monitoring and associated administrative reports. Apparently there is little time for methods development or even proper calibration of certain analytical procedures.

Even within the limited time available the staff have implemented some useful techniques for monitoring plant performance such as the the GC/FID method for purgeable organics. This technique is already valuable for assessing trends in plant performance but could be greatly enhanced by implementing standard quality control/quality assurance practices such as blanks, duplicates, spikes, calibration curves, determining compound recoveries, and external reference standard checks.

The hot water spray "sniffer" installed on the treated water lines at both water treatment plants is a useful tool for assessing plant performance. However at the time I visited the E.L. Smith plant several other chemical operations were being performed in the same room and it was difficult to distinguish the sniffer odor from the extraneous odors in the room. The same problem existed in the Rossdale plant but to a lesser extent. If this device could be isolated from other odors it would be an even more valuable tool. In addition there is no reference against which the odor coming from the sniffer can be compared. When I smelled the sniffer device at the Rossdale plant on March 27, 1986 I detected a moderate earthy odor whereas operators at the plant were listing it as "no odor." If a reference sniffer device spraying "odor-free" water was installed next to the existing apparatus as a point of reference much more subtle odor notes could be observed and all odors could be detected more reliably.

Existing resources in the laboratory could be more fully utilized if there were more manhours available to set up and operate the appropriate analytical methods. The Varian 6000 GC could be used for purge-and-trap analyses; the Hewlett-Packard 5710 GC could be used for organophosphorus pesticides; and the Hewlett-Packard 5830 GC could be used for organochlorine pesticides and FID analysis of extracts (e.g. Closed Loop Stripping extracts). Additional equipment would advance laboratory capabilities. A Dohrmann-Xertex model DC-90 Total Organic Carbon analyzer with purgeable organic carbon attachment(or similar instrument) would be a useful tool for rapid and accurate analyses of the sum total of purgeable organics. This method would be suitable for monitoring plant trends and the purge-and-trap technique could be used at a reduced frequency with greater QA/QC for monitoring specific compounds. A Tekmar model ALS ten unit autosampler(or similar instrument) would allow unattended purge-and-trap operation and thereby allow more quality control samples to be run with the same manpower input. Eventually a benchtop model GCMS would be useful in rapidly tracking and identifying compounds; but this would not be a wise investment until laboratory manpower has been increased such that existing equipment is fully utilized with standard quality assurance practices.

Technical Approach

The changes in general approach and laboratory operations discussed above would provide the framework for taking a sound scientific approach to the water quality and treatment issues facing the City of Edmonton in the future. The basic elements of this suggested approach would be as follows:

Define problem
Define conceptual approach to problem
Develop Quality Assurance plan
Conduct methods development as needed
Conduct monitoring program
Conduct bench scale experiments
Conduct pilot scale experiments
Conduct plant scale tests as feasible
Implement plant modifications
Problem solved!

Of course not all of these steps will be necessary in each case but they will provide a uniform framework for water treatment process staff and laboratory analysts to surmount even the toughest water quality issues.